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TO

ROBERT G TORREY M D

Professor of Medicine
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# FOREWORD

The rapid strides in electrocardiography roentgenology and blood chemistry in the diagnosis treatment and prognosis of heart disease have complicated the picture tremendously from the general practitioner s stand point Unless he is able to sacrifice a number of months for special study in the larger medical centers it is almost impossible for him to follow the speakers at the various medical meetings or to understand the more recent articles on the subject of cardiovascular disease

The author of this book has hit upon a happy solution of this problem By outlining the diagnosis and treatment of such a large number of cases he offers to the general practitioner an example of practically every cardio-vascular problem which he may meet in his practice From this standpoint I believe no more valuable book has appeared

WILLIAM D STROUD

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# PREFACE

Many advances are evident in all fields of modern medicine The vast increase in knowledge in the different branches of clinical science has produced new specialties, some of which have already reached maturity and others are in the process of development The patient should no longer hope that one man can retain sufficient understanding of all branches to be a competent family adviser On the other hand the large gaps that are forming between the physician specializing in various fields and the man in general practice will ultimately react to the detriment of the patient For example, the specialist or research worker who is usually closely associated with a medical school becomes so engrossed in the study of physiologic and pathologic phenomena as well as in the newer methods of diagnosis that he frequently overlooks treatment The general practitioner however becomes so busily engaged in treatment that he has little time to take note of the progress made in the other branches of clinical medicine After all, the family doctor is human and though many other things in the modern world have changed a 24-hour day is still standard for him If in a burst of energy he decides to review heart," he discovers to his dismay a voluminous literature on the subject New terms new concepts even new operative procedures designed for the cardiac patient greet him on every side He becomes discouraged when he finds that the principles guiding management are often buried beneath an avalanche of scientific data or are presented in too meager a fashion to serve as an adequate guide to him at the bedside So it is small wonder that his wanderings in unfamiliar territory soon cease

This small volume attempts to assemble the facts that are most essential in the management of the cardiac patient Physical and laboratory methods of examination that aid in the diagnosis are described and evaluated Emphasis is placed on the present-day recognition of the importance of classifying and treating heart disease according to its etiology and the functional capacity of the patient rather than in the light of the structural defect Material for this book has been collected during the past ten years which have been devoted to the teaching and practice of cardiology These ten years followed seven years in general work I have therefore had little difficulty in arranging the contents to fit the present-day needs of the general practitioner

In choosing the case method of presenting this subject I have tried to show the variations that occur in the different types of heart disease that are commonly met in office and clinic, as a result most of the situations which appear in cardiologic practice are covered For representative cases I have drawn heavily on material presented at Saturday clinics for the third year classes at the Woman's Medical College and to the fourth year

treatment of the different types of heart disease The description of the operative procedures used in hypertension, suppurative pericarditis, aneurysm and effort syndrome have been written by Dr James Lehman Dr Charles Steiner has furnished the description of the injection treatment of angina and Dr Henry D Jump has contributed from his experience in the wiring of aneurysms

Allergic factors of importance in the management of heart disease have been fully covered in a chapter by Dr Richard Kern, Professor of Clinical Medicine at the University of Pennsylvania Dr I S Ravdin, Professor of Research Surgery at the same institution has contributed a surgeon s impression of the relationship between gallbladder disease and the heart

The orthodiagrams the electrocardiograms, and the charts unless otherwise indicated are the work of my secretary and technician Miss Eleanor Lowe I am also indebted to her for typing the manuscript and assisting in the proofreading The roentgenograms are largely from the laboratory of Dr Jacob Vastine Clinical Professor of Roentgenology at the Woman s Medical College

It is a pleasure to express my appreciation to Mr Howard Gosner photographer to the Laboratories of the Philadelphia General Hospital for his excellent photographs of autopsy specimens The drawings in the text have been furnished by Miss Elizabeth Minter and the frontispiece by Mr Maulsby Kimball Jr My obligations to them are obvious

Repetition has been avoided as much as possible to keep the size of the book within the limits of usefulness For this reason the reader must be asked to excuse the frequent references to other sections of the text, if these become annoying at times

The pages that follow do not comprise a complete treatise on cardiology Much material of an experimental and controversial nature has been omitted for it is not my purpose to act as reviewer of a parade of cardiologic fact and theory Neither do I desire to claim originality for all the opinions expressed For this reason a selective bibliography has been added to aid the physician in the further pursuit of a topic of special interest This list may be of some advantage since packet systems established by state and national societies bring the recent literature to the front door of any physician in the country at short notice

Finally it is hoped that the man in the field far removed from our modern medical centers who sees heart disease in its incipiency as well as in its hopeless terminal stages may find this volume a guide to a more intensive study Then, instead of merely reading about the 'increasing incidence of circulatory disease in our country today in books news papers, and medical journals, he may unite with us in an attempt to answer the question 'What can be done about it?

WILLIAM G LEAMAN, Jr

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classes of the same institution during ward walks at the Philadelphia General Hospital I have also included some records of patients from the cardiac clinics of the Woman's College Northeastern and Memorial Hospitals in Philadelphia I have selected from my own office files most of the cases in which home treatment is described Only essential and pertinent data necessary to visualize the clinical picture are presented in these case summaries

The general practitioner is still responsible for the care of the majority of cardiac cases in our country today consequently the methods most useful in home treatment are presented in detail If it appears that small items in the care of the cardiac patient are overemphasized I offer no apology for I have learned from a close study of a number of records that most mistakes are apt to occur at these points For this reason illustrations have been inserted whenever possible to show not only the essential equipment but also the technic for various diagnostic and therapeutic procedures It is hoped that the frequent inclusion of orthodiagrams roentgenograms and electrocardiograms will permit the reader who is unfamiliar with the value of these methods in diagnosis to review them with the case record A knowledge of the gross pathologic changes that take place in the cardiac structure in each of the various types of heart disease is valuable in understanding the physical signs and in planning a regime of treatment Consequently whenever a necropsy was performed on the patient under discussion the photographs have been included In some instances no doubt my opinion as to the proper method of treatment of these cases will differ from the views held by others If so I hope that the beliefs set down here will not be allowed to stand unchallenged

The electrocardiographic method is here to stay It has proved its usefulness at the bedside and is an essential part of the equipment of every hospital and of many practitioners A knowledge of the fundamentals of the subject can no longer be disregarded by the progressive physician The growing interest of the general public in the subject of heart disease and the decreasing cost of the apparatus essential to the practice of cardiology have placed this new diagnostic equipment in an increasing number of Main Street offices from coast to coast However, the usefulness of the electrocardiograph to the physician does not begin the day it arrives from the manufacturer To understand its place in the management of the cardiac patient the physician should study the instrument and become acquainted with its advantages as well as its limitations If ownership is acquired so also is the obligation to learn at least the fundamentals of electrocardiography Without preliminary experience in taking and interpreting the electrocardiogram the equipment alone will add little to the diagnostic skill of the physician Furthermore in untrained and careless hands the electrocardiograph may actually be dangerous if unscientific and unwarranted diagnoses are turned out

With this in mind an attempt has been made to write a description of electrocardiography for the beginner in the simplest terms possible Controversial aspects have been avoided and only the facts essential in the

interpretation of the graphs are presented briefly and in logical sequence Numerous electrocardiograms have also been included with the case records to aid the physician in assessing the value of the method in each instance Little difficulty should be encountered particularly in the understanding of the cardiac arrhythmias, if from the start we train ourselves to think electrocardiographically ' As a richer experience is gained in the laboratory of every-day practice the electrocardiograph will then be seen to be an absolute necessity in fewer instances

I have been slow to adopt some of the recent surgical methods that have been advanced for the treatment of various types of cardiovascular disease In particular, attempts on the part of surgeons to influence the course of essential hypertension I have been guilty at one time or another of labelling as risky empirical and useless As the years have passed however I have seen patients with beginning malignant hypertension, who seemed doomed under my care to a rapid hopeless, downhill course aided by operation to an extent that I cannot in fairness overlook if I am to present here an unbiased view of modern methods of therapy Therefore, when surgical treatment is described in the following pages I have stepped aside and invited my surgical colleagues to continue the discussion and present their cases The reader must realize that these newer procedures that attack the glands of internal secretion and the nerves of the vasomotor system are far from being accepted methods in all instances I must admit that our surgeons to date have a far better record in bringing a large measure of relief to selected cases of malignant hypertension than I can claim This fact has been too clearly evident to me as I have reviewed a number of case histories

Success in the treatment of heart disease in our hospital out patient clinics is incomplete without a good social service department What is contributed by these workers often spells the difference between a discouraged misfit in the business world and a well adjusted patient living within the limits of his myocardial reserve No treatise on the management of the cardiac patient would be adequate without a description of the aims and methods of the social worker I am fortunate in being able to include a special chapter on this aspect of the treatment of heart disease written by Miss Helen Heikes secretary of the Philadelphia Heart Association assisted by Miss Olga Tattersfield of the Social Service Department of the Woman s College Hospital

The arrangement of this material with the thought of publication in mind was first discussed with Dr William D Stroud and Dr Robert L Levy in October 1938 at the time of the 88th Annual Session of the Medical Society of the State of Pennsylvania If the delay had not been so great in an overcrowded Scranton restaurant during one lunch hour I would never have had the advantage of such good counsel and this volume would not have reached the publisher in its present form—if indeed, it would have reached him at all

The help rendered by my colleagues on the Faculty of the Woman s Medical College is evident as their names appear in the discussion of the

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# 1

# APPROACH TO THERAPY

## INTRODUCTION

We are witnessing great advances in therapy today Sulfanilamide and its derivatives are conquering infections of various types newer forms of insulin have been perfected to bring greater comfort to the diabetic, in the treatment of the psychoses gratifying results have followed the use of newer remedies Pyrexial therapy the sex hormones the vitamins a variety of safer anesthetics reflect many advances in other fields The treatment of diseases of the heart has not lagged behind Today more can be done for the cardiac invalid than was possible a decade ago A better understanding of the mechanism of heart failure a clearer concept of the use of digitalis and the introduction of newer diuretics are only a few of the factors that enable the modern physician to bring a degree of relief to cardiac patients not dreamed of at the beginning of the present century

The appearance on the scene of many laboratory methods of precision characterizes the modern era While these attract a considerable amount of the attention of research investigators they should not be allowed to replace the older methods of physical examination We must realize that even the foremost among these electrocardiography and roentgenography taken together cannot furnish the amount of information that may be obtained from a carefully taken history and a thorough physical examination All laboratory methods have great pitfalls for the ones who in the hurry that is typical of modern times lean upon them too heavily Laboratory methods of precision are only aids to be sought in establishing a diagnosis that is suggested after all the facts in the case have been collected recorded and fully evaluated If this procedure is always followed often the diagnosis will stand out so clearly that there will be scant need for a number of expensive laboratory tests For example a good clinician will not permit a patient who has a precordial friction rub following an attack of agonizing chest pain to be moved any distance for the sole purpose of obtaining an electrocardiogram Nor will a roentgen examination add a great deal to the patient s diagnosis when the Wassermann reaction is positive the pulse is of the Corrigan type and the apex beat is visible in the midaxillary line

In some instances however after all available data are carefully considered laboratory procedures will be necessary to help clarify a diagnosis The Wassermann for example is a great help in making a decision as to therapy in a patient of 40 whose only lesion appears to be aortic regurgitation and whose past history is negative for rheumatism and syphilis In

some cases of suspected coronary occlusion the entire examination of the patient may be negative and the diagnosis will be revealed only in the chest leads of the electrocardiogram Often the electrocardiogram may be the sole evidence of the presence of acute myocarditis However the instances are few where the laboratory acts as the cornerstone in diagnosis An attempt should be made in every case to arrive at the cardiac diagnosis unaided for in this way the ability of the physician to diagnose heart disease will increase with his experience and in emergencies he will find that he can direct treatment efficiently when laboratory aid is unavailable

## THE HISTORY

The best approach to therapy is a brief consideration of the methods commonly employed in arriving at a diagnosis of heart disease Careful history taking remains the most important but often the most neglected feature of the entire examination It should never be relegated to an assistant for impressions that may be essential to diagnosis are made in taking the history Furthermore in certain types of heart disease the diagnosis can be made on the history alone I make a rule to take a long history but to write a short one

**The Patient s Description** It is wise to permit the patient to tell his own story and allow him plenty of time to review it completely I share the patient s dislike for any system that entails the routine asking of a large number of questions by an office assistant who fills in a printed form, but perhaps this is only a personal expression of rebellion against the present day system of regimentation In any event stereotyped questions usually receive the same type of answers and although they may be very useful in subsequent research this system deprives the physician of the opportunity of witnessing in an informal friendly interview the unfolding of the patient s personality with the story of his symptoms Frequent interruptions confuse and irritate the patient and usually inhibit the growth of confidence in the physician that is so essential at the first interview Let the patient talk It is an easy matter to jot down roughly the important features of the narrative and either dictate or rewrite the history later Of course this freedom in the telling should not be allowed some patients who deal at length with multiple and irrelevant issues However even in these cases much can be learned concerning the probable presence or absence of heart disease if the physician trains himself to be a good listener Further amplification of important symptoms lightly touched upon by the patient may be obtained by asking a few direct questions at the end of the interview As many as possible of the adjectives used by the patient in describing an important symptom should be recorded using quotation marks The description of the complaint at a subsequent visit by the use of the patient s own phrases establishes confidence in the physician This is particularly true in regard to the variety of expressions used by patients in describing chest sensations Many are fearful of the diagnosis of angina yet desire

to give the physician the most accurate account possible of the chest symptom consequently their descriptions may be rich in clarifying similes

**The age of the patient** is a most important fact in itself in cardio vascular diagnosis Certain types of heart disease are more frequent in certain age groups For example under two years of age it is unusual to meet any form of heart disease other than congenital After this age and during the first decade of life we have to consider rheumatic carditis plus congenital heart disease From this time until 40 rheumatic heart disease dominates After 40 cardiovascular syphilis becomes a consideration and between 40 and 60 coronary and hypertensive cardiovascular diseases predominate while congenital defects are very rare and the rheumatic lesions very infrequent From 60 to 80 coronary or arteriosclerotic and hypertensive varieties hold the stage these types are usually present as the final curtain falls

**The sex** of the patient is an important consideration in etiology For example it is a statistical fact that the coronary and syphilitic types of heart disease occur with greater frequency among males Calcific aortic stenosis likewise affects men more often than women Rheumatic and hypertensive heart disease however seem to claim an equal number of victims from both sexes Thyrotoxic heart disease on the other hand is more prevalent among women

**Hereditary Factors** It is very important to inquire carefully into the causes of death of all near relatives From the frequency with which I have encountered rheumatic hypertensive and coronary artery disease in members of the same family it appears that the incidence of these diseases is influenced by hereditary factors A positive family history always adds weight to the diagnosis of angina when the cause of an atypical chest pain in an active business man in middle life is considered

**The past medical history** has a direct bearing on the type of heart disease that may be present If several typical attacks of rheumatic arthritis occurred in childhood evidence enough is at hand to suspect this type of cardiac involvement Chorea may be viewed in the same light Growing pains quinsy tonsillitis may or may not be rheumatic in nature but provide evidence that should be weighed carefully "Usual diseases of childhood" is a routine meaningless phrase that appears on nearly all hospital records Diseases if recalled should always be listed by name Diphtheria and scarlet fever usually have no effect in later years on the cardiac status of the patient

**Syphilis** Inquiry as to the presence or absence of a syphilitic lesion should always be made toward the end of the interview This question is a very important one particularly in patients in middle life who exhibit signs of aortic disease It should never be asked in a tone that invites denial If the patient is a woman a visible primary lesion may not have been present In this event clues will have to be sought carefully during the subsequent physical examination

### Cardiac Symptoms

In each patient it is important to attempt to establish accurately the date of the first appearance of cardiac symptoms A brief summary of the progress of each symptom to the date of the examination should follow Cardiac symptoms of importance are chest pain abnormalities of breathing or dyspnea and palpitation They are described differently by each patient because the nervous mechanism that detects and interprets them is a factor that shows considerable individual variation In no case will the symptoms be directly proportional to the amount of structural change producing them Moreover they may all be present in an oversensitive individual in the absence of any type of organic cardiac disease They *may appear at one time and not at another for example, when fatigue* enters the picture and lowers the patient s threshold of perception

Chest pain is the most important of all cardiac symptoms Care should be exercised in its description in the history for in a few instances it may be the only positive finding in the entire case record If the chest pain or oppression is transient and definitely related to exertion, and relieved by rest and nitroglycerine, the diagnosis of coronary disease is at once suggested Pain having the same qualities and significance may be experienced in regions distant from the heart—for example in the neck jaw shoulder, arms or even the abdomen

The pain that accompanies occlusion of a coronary artery or any of its branches usually can be differentiated clinically from the pain of angina When occlusion occurs, the transient pain of angina gives way to a prolonged agonizing type of pain that is accompanied generally by marked changes in the appearance of the patient The pallor sweating and shock that accompany occlusion are important points to elicit and record if as is usually the case the person who witnessed the attack accompanies the patient to the office

Chest pain may be a feature of other types of heart disease A dull precordial ache is complained of by patients suffering from effort syndrome It often accompanies the overactive heart of thyrotoxicosis and may be present in patients who have cardiac hypertrophy from any cause but particularly if it is of the hypertensive type The nervous system of some patients is unfortunately very closely in tune with the cardiac action Consequently irregularities such as frequently recurring premature beats will be readily detected and may produce painful stimuli At times they may be the cause of knife like pains in the cardiac area Acute pericarditis may produce a precordial pain that is increased by respiration owing to the extension of the inflammatory process to the outer layer of the pericardium and thence to the pleura or diaphragm If effusion follows the pain may disappear entirely or it may give way to a dull precordial ache

Aortic aneurysm by its growth and erosion is responsible for a severe and constant type of chest pain The pain produced by dissection of an

aortic aneurysm is perhaps the most excruciating variety experienced by man It is sudden in onset of maximum intensity from the start and is referred to the front or side of the chest or to the abdomen Patients often describe it as tearing It may however be confused with the pain of coronary occlusion in some cases (page 323)

**Dyspnea** has many causes but there are usually three types commonly described in relation to diseases of the heart In early cardiac failure the patient becomes conscious of the respiratory act As the lungs become engorged following left ventricular failure reflexes to the respiratory center usually increase the rate of breathing As failure progresses dyspnea may be present at complete bed rest We use the term orthopnea when the dyspnea is extreme and the respiratory act demands the patient's whole attention and much of his failing strength The patient with orthopnea is forced to sit up in bed in order to employ the accessory muscles of respiration as well as the effects of gravity to relieve the over loaded pulmonary circulation

An increasing degree of dyspnea usually parallels the loss of cardiac reserve and is valuable in estimating prognosis in any patient under treat ment The amount of breathlessness that follows the same task assigned at each visit should always be carefully observed and recorded

**Cardiac Asthma** The sudden attack of dyspnea that often follows acute failure of the left side of the heart during sleep when the head and chest are low is known as paroxysmal cardiac dyspnea or cardiac asthma These distressing episodes frequently occur toward the end of life when the types of heart disease that place the extra load on the left ventricle are common It is important to record the date of onset of these seizures in the history

**Cheyne Stokes Respiration** In the advanced stages of some types of heart disease poor cerebral circulation following sclerosis and declining cardiac reserve markedly affects the respiratory center Periods of apnea or cessation of breathing alternate with periods of increased breathing or hyperpnea producing the well known clinical picture of Cheyne Stokes respiration Relatives who are constantly in attendance often give accurate descriptions of this symptom which when marked indicates a poor prognosis

**Palpitation** or consciousness of the beating of the heart is a very common but a much less important complaint The patient usually classes several types of cardiac happenings under this heading which must be carefully sorted out when the history is taken The occasional disturbances of the heart's rhythm caused by extrasystoles reach consciousness and are classed as palpitation The sudden onset of paroxysms of fibrillation flutter or tachycardia are usually felt by the patient and also described under the heading of palpitation Consequently when palpitation is the symptom question the patient in detail to bring out the exact nature of the disorder Palpitation is a very common symptom in effort syndrome and thyrotoxicosis Many of the patients complaining of palpitation will be found

to have no underlying cardiac disease Consciousness of the heart's action is a very common complaint among those suffering from anxiety neurosi

**Syncope vertigo and faintness** may all be directly associated with some form of heart disease Different degrees of cerebral anemia due to slowing of the pulse or a fall in blood pressure or both may be responsible In patients with aortic regurgitation and stenosis and varying degrees of heart block vertigo and syncope are important symptoms to elicit Occasionally sudden occlusion of a coronary artery may cause syncope Very rapid cardiac rates when coronary filling is incomplete may be followed by a loss of consciousness On the other hand syncope may occur after any mild exertion excitement or emotion in a susceptible person The mechanism here is either vagal or vasomotor or a combination of these two elements

Finally many gastro intestinal symptoms (anorexia gas nausea upper abdominal pain) may be complained of by the cardiac patient They may arise in the course of coronary disease or may accompany congestive failure Hoarseness is caused in rare cases by the presence of a large left atrium secondary to mitral stenosis or may follow pressure of an aortic aneurysm on the recurrent laryngeal nerve Cough and hemoptysis are not uncommon symptoms associated with the pulmonary congestion that accompanies failure of either ventricle Pulmonary symptoms often predominate in the later stages of mitral disease

## THE EXAMINATION OF THE PATIENT

The physical examination of every patient begins as soon as he enters the physician's office The speech the manner and the emotions as the history is given reflect at once the patient's mental background These impressions should always be noted for they are very helpful in the evaluation of the cardiac symptoms In making complete physical examinations it is wise for the beginner to adopt an orderly system and carefully record all findings With experience and constant practice the physical status of a patient may be determined very quickly and only the positive findings need be recorded Confining the physical examination to the chest will lead to grave errors in diagnosis since signs in other regions at a distance from the heart may be most important in the discovery of the kind of heart disease present

**The type of breathing** and the respiratory rate are important observations to make Orthopnea seizures of cardiac asthma breathing of the Cheyne Stokes type all give warning of grave danger ahead whereas the patient who is frequently compelled to pause to take an unusually deep breath (sighing type of respiration) is usually giving a valuable hint that suggests the absence of serious cardiac disease

**The color of the face and lips** as well as the distribution of this color should be noted In patients with mitral disease purplish red patches of color (malar flush) may be observed Marked pallor of the face may be

seen in patients suffering from coronary sclerosis and at times in patients with aortic disease

**Cyanosis** is due to change in the character of the circulating blood appearing when the capillary blood contains more than five per cent of reduced hemoglobin It is seen in 35 to 50 per cent of the cases of congenital heart disease and occurs when over one third of the output of the heart is shunted from the right to the left side without passage through the lungs Advanced mitral lesions interfere with pulmonary circulation and cyanosis may follow improper oxygenation Hydrothorax and edema of the lungs both interfere with proper pulmonary aeration On the other hand the extreme cyanosis found in patients with pulmonary sclerosis



Fig 1 The pitting edema of congestive cardiac failure

(black cardiacs) is often caused by the pulmonary disease entirely and not by any cardiac defect Heart failure from any cause may produce cyanosis since the slow circulatory rate in this condition produces capillary stagnation and greater reduction in the hemoglobin

**Edema** is an important sign in heart disease It may develop suddenly or slowly and may vary from a slight pitting of the ankles appearing only at night to a generalized anasarca As heart failure develops edema is first noted in the lower extremities toward the end of the day while in patients confined to bed it may appear over the sacral region The edema of the feet that accompanies early cardiac failure tends to disappear after a night s rest and to recur at the end of the next day The condition may increase until pitting (Fig 1) is present at all times Fluid may then

accumulate in the pleural cavity (hydrothorax), in the abdomen (ascites) (Fig 2) and finally become generalized (anasarca)

Cardiac edema is a symptom of hypertension in the capillary bed and *varies with the venous pressure* It accompanies right heart failure and conditions that prevent proper diastolic filling (chronic constrictive pericarditis) Left heart failure and shock are not accompanied ordinarily by edema because they produce a diminution of the blood flow that is not associated with increase in capillary pressure

Edema of the feet may be present in renal disease Such edema is usually worse in the morning and is associated with marked swelling of the face In the later months of pregnancy edema of the feet may be present owing

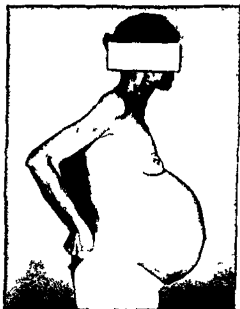


FIG 2 Ascites accompanying congestive cardiac failure

to uterine pressure on the large veins (page 445) Other abdominal tumors may also cause edema and lead the physician to suspect the presence of cardiac disease Severe grades of anemia can produce all the symptoms of cardiac disease, including edema Myxedema (see Fig 142) will rarely be mistaken for the edema of cardiac failure because of the thickening of the *skin that accompanies this deficiency state and the failure of the extremities* to show pitting on pressure Rarely Milroy s disease (persistent hereditary trophedema) may simulate cardiac edema but its presence in several generations of the family and the demarcation between swollen and nonswollen parts serve to make the distinction evident In beriberi of the wet type (see Fig 163) a generalized edema may occur In this disease an accumulation of fluid in the heart muscle has also been described

## Inspection

**Eyes** In the physical examination of the patient inspection of the eye grounds is commonly omitted This is unfortunate when we consider the amount of information that may be obtained by one familiar with the use of an ophthalmoscope Many times the mark of a previously existing hypertension will be seen in the retina and this will support the diagnosis of hypertensive cardiovascular disease if the blood pressure happens to be low at the time of the examination The recent use of slit lamp inspection of the small capillaries of the conjunctiva has shown that these vessels exhibit changes in their structure suggestive of hypertension even earlier than the arterioles of the retina Gross inspection of the conjunctiva for petechial hemorrhages is also important in cases where subacute bacterial endocarditis is suspected

The presence of exophthalmos and related eye signs of hyperthyroidism may be important observations in explaining the cause of a persistent tachycardia The pupillary reflex to light as well as irregularity of the pupils may lend support to a diagnosis of syphilis It is well to keep in mind the fact that over 50 per cent of the cases of cardiovascular syphilis are complicated by neurosyphilis

**Infection** about the margins of the teeth should be carefully noted as well as all nonvital and abscessed teeth The pharynx should be inspected for infected lymph follicles and the tonsils carefully examined for evidence of hypertrophy or infection The condition of the anterior pillars is important when considering tonsillar infection and the presence of tags may have a decided influence in favoring the recurrence of the upper respiratory infections that are so dangerous in rheumatic subjects The lymph nodes draining all these structures should be palpated carefully

**Neck** Examination of the neck furnishes many important clues Careful inspection of the vessels may permit the observer to diagnose the nature of the cardiac arrhythmias For example detection of abnormal venous pulsations in the jugular veins of a patient with a ventricular rate of 40 may clinch the diagnosis of heart block while a more rapid series of waves may be seen in the jugulars in cases of auricular flutter The engorgement and pulsation of the jugulars in cases of congestive failure and chronic constrictive pericarditis (see Fig 82) signify delay in the return of the venous blood to the heart and increase in the pressure on the venous side Consequently it is evident that a close study of the neck veins is a great aid in the diagnosis and treatment of the cardiac patient

Increased pulsations in the carotid arteries occur in patients suffering from aortic regurgitation hypertension and aneurysm In the latter instance tracheal tug may also be palpable The region of the carotid sinus is important since pressure here in hypersensitive patients may cause syncope (page 378)

**Chest** Careful inspection of the chest of the cardiac patient will richly reward the examiner Slight but definite bulging of the left side of the

precordium (Fig 3) usually indicates cardiac disease in youth before the calcification of the costal cartilages consequently either rheumatic heart disease or a congenital defect should be suspected Kyphosis scoliosis and at times depressed sternum or funnel chest are important clues since they may so compress and displace the heart as to interfere seriously with its

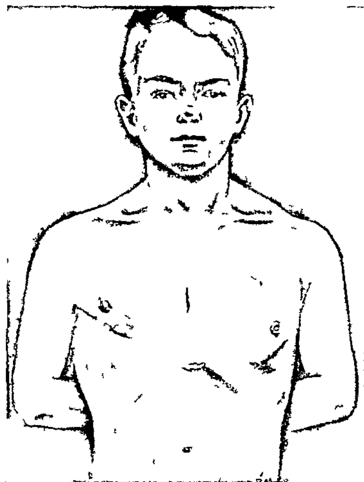


Fig 3 Precordial prominence in a child caused by a greatly enlarged heart

action In thin chested individuals an apex beat visible in the sixth inter space beyond the midclavicular line is evidence of enlargement of the left ventricle Right ventricular enlargement is suggested by pulsations in the third and fourth interspaces to the left of the sternum Retraction of the whole thoracic wall about the region of the cardiac apex is suggestive of an adherent pericardium Although rare sights today pulsating masses on the anterior thoracic wall suggest aneurysm of the aorta (see Fig 91)

## Palpation

Thrills palpable over any area of the chest are important indications of narrowing valvular orifices They are best appreciated by light palpation A diastolic thrill over the region of the cardiac apex suggests mitral stenosis When a rough systolic murmur is heard over the second and third interspaces to the right of the sternum the presence of a systolic thrill in this region will add considerable weight to the diagnosis of aortic stenosis Rarely an aortic aneurysm will produce a systolic thrill in the same area A thrill over the region of the third interspace to the left of the sternum points to a congenital defect of the interventricular septum When a thrill is felt over the pulmonary valve area it is generally associated with stenosis of this valve and is usually congenital in origin When a continuous thrill is present in this area accompanied by loud systolic and diastolic murmurs a patent ductus arteriosus may be suspected

## Percussion

In spite of many opinions to the contrary in literature I still believe that percussion is a worth while method for estimating cardiac size However the necessary degree of skill in this branch of physical diagnosis can only be acquired by constant daily practice In the majority of patients the size of the heart can be approximately obtained without resorting to more accurate roentgen methods Corvisart [3, 4] said

> Percussion of the thorax is the best touchstone we have to investigate or at least clarify our knowledge concerning many of the lesions of this cavity By its means we can demonstrate an increase in the volume of the heart one may even go so far as to estimate the severity of the lesion it cannot be used too frequently by means of tactile sense a deaf person may make use of percussion

**The left cardiac border** should be percussed first The change of note in normal patients will be found to occur 8 to 9 cm to the left of the mid sternal line in the fifth interspace When this border is determined the midclavicular line can be indicated on the chest surface and the relationship noted The remainder of the left border of the heart can then be completed percussing in each interspace from axilla to sternum until a change in note from resonance to dulness is heard Care should be taken in the third interspace for lesions affecting the pulmonary artery and pulmonary conus (mitral stenosis patent ductus arteriosus) may cause an increase in the diameter at this level A change may be noted by careful percussion even in cases where the apex beat is in a normal position

**Right Border** In the percussion of the right border of cardiac dulness more difficulty is usually encountered This border follows the right sternal margin to the fourth or fifth interspace where it bulges slightly outward and with care can be percussed for a maximum distance of 5 cm from the midsternal line Percussion over the sternum usually sends the whole

structure into vibration and in the presence of this resonance localization of the heart border is impossible Likewise fine differences in the percussion note here are scarcely appreciated in patients with emphysema and obesity In determining cardiac size in these cases the roentgen methods have their greatest usefulness

If in the course of the examination, I fail to locate the apex beat I always attempt to outline the heart by percussion before proceeding with fluoroscopy The opportunity of checking the result by an orthodiagram keeps alive my belief in the value of ordinary percussion This special training

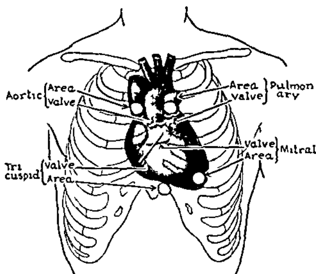



FIG 4 The valve areas and the anatomic location of the heart valves

of the senses of touch and hearing proves a valuable asset at the bedside far from the roentgen laboratory The determination of heart size is the most important single feature of the examination of the cardiac patient, since an enlarged heart is usually a diseased heart

**Aorta** While I believe that the heart dulls somewhat the ringing resonance sufficient to permit its outline by percussion I do not hold the same opinion in regard to the great vessels at the base when they are normal in size If the aorta is inspected in the cadaver it will be seen to arch in a direction away from the chest wall In addition the vessels at the base of the heart are surrounded by lung tissue Consequently unless the aorta is greatly enlarged I doubt my ability to indicate its diameters with any degree of accuracy by percussing the anterior chest wall

## AUSCULTATION

In auscultation of the heart it is important to examine the patient both in the upright and recumbent positions Often certain types of murmurs will be heard only after exercise when the patient is lying on the left side

Auscultation should be carried out over all the cardiac valve areas (Fig 4) I prefer to use the combination type of stethoscope (Fig 5) since murmurs not audible with the bell type of chest piece may at times be clearly heard when the Bowles attachment is used The faint murmur in early mitral stenosis usually is heard best when the bell of the stethoscope is used, while the soft diastolic murmur of aortic insufficiency is detected best by the Bowles attachment

## HEART SOUNDS AND MURMURS

In cardiac auscultation it is important to record the rate rhythm, character and intensity of the heart sounds and the quality area of distribution and the place in the cardiac cycle of any murmurs present Two sounds are normally heard over the heart area The first sound is louder and lower in pitch than the second sound Over the base (aortic and pulmonary areas) the second sound is louder and higher in pitch than the first The aortic second sound in adults usually equals the pulmonic second sound in intensity The closure of the tricuspid and mitral valves and the contraction of the ventricular muscle are the two elements that combine to make up the first heart sound The second sound is produced by the closure of the aortic and pulmonic valves at the end of cardiac systole

**The first sound** of the heart at the apex is normally accentuated after exercise and during fever Abnormal accentuation, however may accompany mitral stenosis, effort syndrome thyrotoxicosis and the paroxysmal tachycardias

Decrease in intensity of the first sound of the heart may be normal in the presence of obesity and emphysema Decrease from day to day may occur if there is an accumulating pericardial effusion or it may accompany infarction of a large section of the cardiac wall In peripheral circulatory collapse where there is a diminished venous return to the heart (shock hemorrhage) decrease in the intensity of the heart sounds may be noted Variations in the intensity of the heart sounds takes place in the cardiac arrhythmias (premature beats heart block auricular fibrillation)

**The aortic second sound** is louder than the pulmonic second sound in individuals over 60 but hypertension in the systemic circulation may cause this accentuation to occur at any age Degenerative changes in the aortic valve (arteriosclerosis and syphilis) may sometimes change the character of the aortic second sound in the absence of hypertension

Aortic and mitral stenosis by decreasing the amount of blood ejected into the aorta at each systole may be accompanied by diminution of the aortic second sound Disease of the valve cusps preventing proper closure may be another factor while sudden left ventricular failure produces the same effect Premature beats may have just sufficient force to open the valves in which event the amount of blood received by the aorta is small and the aortic second sound is weak Many premature beats do not open the pulmonic and aortic valves at all consequently the second sound is

absent In hypotension in certain anemias in prolonged fevers, or following peripheral failure there may be a diminution of the aortic second sound

**Accentuation of the pulmonic second sound** indicates increase in the pressure in the lesser or pulmonic circuit This may be secondary to a left ventricular failure or mechanical defect of the mitral valves, or it may point to a pulmonary hypertension that is seen in some conditions for example tuberculosis emphysema or pulmonary sclerosis that are forerunners of a right sided heart failure

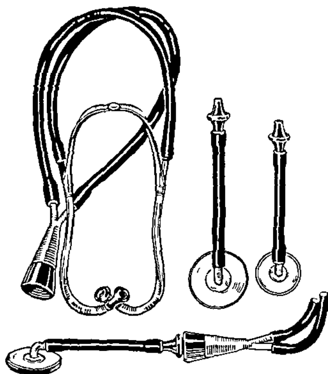


Fig 5 Pilling Bowles combination stethoscope with diaphragm and Ford chest pieces

A diminished pulmonic second sound may be a danger signal of a failing right heart Likewise in the presence of an active stenosing process in the pulmonary valve leaflets the pulmonic second sound may gradually diminish until it disappears Additional signs over this area that should be searched for to confirm this impression are a rough systolic murmur and a palpable thrill

**Splitting of the heart sounds** occurs in health, many times it is related to respiration Splitting of the first heart sound likewise occurs in patients suffering from heart disease for example in intraventricular or bundle-branch block where the ventricles receive their impulses at different times and consequently contract asynchronously (page 623) A splitting of

the second sound at the base of the heart is more common and is caused by asynchronous closure of the aortic and pulmonary valves It may be present normally during certain phases of respiration or it may follow bundle branch block or premature ventricular beat It may accompany either pulmonic or systemic hypertension

**Gallop** In children and young adults a third heart sound may be heard between the apex beat and the left sternal border in the fifth interspace This is a normal finding but at times it may be impossible by auscultation to differentiate it from the extra sound that makes up a gallop rhythm In some cases the clinical findings serve to make the distinction

The term gallop applied to this finding is a good one and should be retained because of its resemblance, especially when the heart rate is above 100 to the sound of the hoof beats of a galloping horse Potain originally described three varieties of gallop rhythm protodiastolic (early diastolic), mesodiastolic and the presystolic (late diastolic) Clinically only two types are important The more common presystolic type presumably is caused by an audible auricular contraction and never occurs in its absence The protodiastolic gallop is a sound that occurs approximately 0 12 to 0 20 second after the beginning of the second sound (Wolferth and Margolies) Gallop sounds are heard best with the patient recumbent and are variable appearing at one examination only to be absent at the next They are altered in the position they occupy in the cardiac cycle and in intensity by changes in the cardiac rate [38] It is well worth while to study these sounds for they often appear with cardiac failure to disappear again as the patient improves following successful therapy They may be produced by either right or left sided failure and are supposedly the result of heightened intra auricular pressure causing the onrushing blood in diastole to strike the weakened ventricular wall with more than the usual force

**Precordial Friction Sounds** The recognition of precordial friction sounds may be helpful in diagnosis and therapy The appearance of a precordial friction rub early in the course of some cases of rheumatic infection may be important in differential diagnosis Friction rubs appearing in patients of older age groups following episodes of chest pain suggest anterior coronary occlusion The pericardial friction is produced by an area of acute pericarditis and is usually a harsh leathery to-and fro sound that coincides with the heart sound and in consequence is independent of respiratory movements In some instances the friction rub may occupy systole alone in which event it may be difficult to distinguish from a murmur However friction rubs change from day to day They likewise may alter their character with firmer pressure of the stethoscope bell or with change in position of the patient On auscultation their point of origin seems to be nearer the ear than the underlying heart sounds which at times are blotted out by the loudness and harshness of the friction

**Clinical Significance of Murmurs** Laennec who was first to describe cardiac murmurs in 1819 believed that they were always produced by organic change in the valve leaflets Later at autopsies he observed normal

valves in patients who had loud murmurs during life This caused him to take the opposite view and he stated that murmurs were of no value in diagnosis Cardiac murmurs may prove to be just as confusing to the physician of today if more attention is focused on them and less on the patient as a whole Murmurs comprise only one feature of the examination and should always be interpreted in the light of other findings The presence of murmurs does not always mean heart disease For example if a loud systolic murmur heard over the pulmonary area in recumbency in a young person is the only finding on physical examination and if it disappears or becomes less intense when the patient assumes the upright position no heart disease is present No treatment for the heart is needed If the patient has been previously informed about the finding and the word 'murmur has become fixed in his mind the task to convince him that he has a normal heart is not an easy one Many women during pregnancy develop systolic pulmonary murmurs that should occasion no alarm concerning the integrity of the cardiovascular system since they usually disappear following delivery Systolic murmurs at the apex in the absence of cardiac enlargement or previous history of rheumatic infection may be viewed in a similar light

**Timing** Murmurs are either systolic or diastolic in time It is a matter of primary importance to establish their place in the cardiac cycle otherwise errors in diagnosis are apt to occur Many physicians as a result of long experience never have to give much thought to the timing of murmurs Mitral stenosis for example is recognized as soon as heard by its characteristic low pitched presystolic rumble In similar manner we learn to know which dog in the neighborhood is keeping us awake at night by the pitch and other qualities of the bark Experienced examiners often depend on the rhythm of the heart to time the murmur However in the presence of an arrhythmia or with rapid pulse rates this method may fail It is far safer to time the murmur by watching or palpating the apex beat or the carotid artery Riesman was the first to point out a very useful method for timing accurately an apical murmur this method he referred to as transdigital auscultation [313] I have found his suggestion helpful in teaching undergraduates to time murmurs during their first days on the medical wards The following description of the method has been furnished by Dr Riesman

> It is necessary to employ a diaphragm stethoscope and not one of the bell type The index or middle finger is flexed at a right angle and its tip is placed directly over the apex beat. The stethoscope is then laid on the horizontal phalanx of the flexed finger at the angle (Fig 6) It will be found that murmurs may be heard nearly as well through the finger as when the stethoscope is placed directly on the chest and since auscultation and palpation are performed at the same place it is obvious that one can tell readily whether a murmur occurs before the finger is lifted, synchronously

with the lifting or afterward The method also serves well for eliciting the Duroziez murmur in cases of aortic insufficiency This murmur really a double murmur, is heard in the femoral artery when pressure is made on it with a stethoscope just below Poupart s ligament Sometimes the pressure is distinctly painful at others the stethoscope disk is too large for making satisfactory compression If the artery is not properly compressed only one sound is heard By transdigital auscultation, the drawbacks just cited are obviated the to and fro murmur can be readily brought out by regulated pressure with the tip of the finger

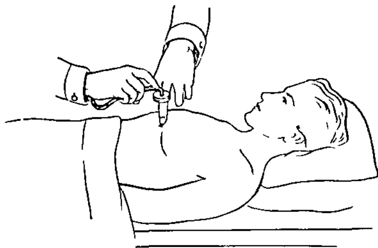


Fig 6 Transdigital auscultation

A murmur is systolic when it occurs between the beginning of the first sound and the beginning of the second, and it is diastolic if it occurs in the longer pause between the second sound and the succeeding first sound A murmur heard in late diastole and continuing up to the succeeding first sound is spoken of as presystolic The heart sound may be replaced by a murmur or both heart sound and murmur may be heard Diastolic murmurs are more serious than systolic murmurs and short murmurs heard with difficulty may be much more important in indicating serious cardiac disease than long and loud murmurs that are readily detected For example the earliest murmur that appears in mitral stenosis is a very short mid diastolic type that often is heard only after exercise with the patient lying on the left side The earliest sign in aortic regurgitation may be a very faint diastolic murmur heard in the aortic area or along the left sternal border

**Determining Site of Origin** It is important to determine the valve area where the murmur has its greatest intensity Murmurs having their

origin in the mitral valve are best heard in the region of the apex beat murmurs arising from the pulmonary valve over the second interspace to the left of the sternum while those having their origin in the aortic valve are heard over the second interspace to the right of the sternum In the latter instance an exception to this rule should be noted In some cases of rheumatic aortic regurgitation especially in children the diastolic murmur is heard best along the left sternal border and may appear to be most intense over the third or fourth interspace close to the sternum

Where a murmur is heard with equal intensity over two areas it may be hard to determine its exact point of origin However if the murmur decreases in intensity as the stethoscope moves away from one valve area toward another the murmur probably originates at the first area If on the other hand the murmur, after diminishing again increases in intensity, as another valve area is approached separate murmurs are probably present The likelihood is even greater if the pitch and intensity of the second murmur differ from the one originally heard

The intensity of any murmur is no criterion of the seriousness of the lesion producing it A small amount of regurgitation at the mitral orifice may cause a loud murmur while a more serious leak may produce a murmur quite difficult to detect or no murmur may be heard at all At times in patients under constant observation if a change can be demonstrated in the quality of the murmur from day to day this may indicate the progression of a lesion of the valvular structure for example, the growth of vegetations in subacute bacterial endocarditis

It is well to adopt some uniform system of terminology in referring to the quality intensity and duration of both heart sounds and murmurs This avoids the use of many indefinite terms that only serve to reveal the uncertainty that exists in the mind of the examiner In Table I the terms in larger type (e g FAINT) are recommended by the American Heart Association for routine use [81] Those in smaller type (e g Weak) are regarded as unsatisfactory (although permissible) synonyms

Some murmurs may be accentuated by exercise and disappear when the patient rests *This is particularly true of the presystolic murmur of early* mitral stenosis Hence a good rule to follow if no murmurs are heard is to auscult again in recumbency after mild exercise Murmurs are better transmitted at the end of expiration when less air is in the lungs In fact faint diastolic aortic murmurs may be detected only at the end of expiration with the patient leaning forward

Cardiorespiratory murmurs usually heard over the lung margins should be easily detected for they are always systolic and are always influenced by respiration Harmless or functional murmurs are most common over the second interspace to the left of the sternum In severe anemia and hyperthyroidism systolic functional murmurs are not uncommon

The intensity of any murmur depends upon the velocity of the blood stream at the moment as well as upon the diameter of the valve orifice producing the murmur The intensity determines how far from the heart the

TABLE 1

TERMS TO BE USED IN DESCRIBING HEART SOUNDS AND MURMURS

| INTENSITY | PITCH | QUALITY | DURATION | TIME |
|---|---|---|---|---|
| *Heart Sounds* | | | | |
| NORMAL | | NORMAL | NORMAL | |
| FAINT | | SHARP | SHORT | |
| Weak | | Snapping | | |
| Distant | | Valvular | | |
| Muffled | | | | |
| LOUD | | BOOMING | PROLONGED | |
| Accentuated | | Muscular | | |
| Increased | | | | |
| ABSENT | | SPLIT | | |
| Replaced by a murmur | | REDUPLICATED | | |
| | | RINGING | | |
| | | Metallic | | |
| | | Bell like | | |
| | | Tambour | | |
| | | Hollow | | |
| *Murmurs* | | | | |
| FAINT | HIGH | BLOWING | SHORT | SYSTOLIC |
| Soft | | | | |
| | MEDIUM | HARSH | MODERATE | EARLY SYSTOLIC |
| | | Rough | | |
| | | Coarse | | |
| LOUD | LOW | MUSICAL | LONG | LATE SYSTOLIC |
| | | RUMBLING | | DIASTOLIC |
| | | CRESCENDO | | EARLY DIASTOLIC |
| | | DECRESCENDO | | MID DIASTOLIC |
| | | | | PRESYSTOLIC (Late Diastolic) |

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murmur may be heard. Each murmur usually has a characteristic area of distribution depending upon the nature of the structures that transmit the sound. For example, the systolic murmur of aortic stenosis is heard in the carotid arteries, while the regurgitant murmur often associated with this lesion is heard best along the left sternal border, which is the direction taken in diastole by the regurgitant column of blood. The murmur of mitral insufficiency is transmitted to the cardiac apex because the structures that transmit the sound come in contact with the chest wall in this area. The column of the papillary muscle plays a large part in the transmission of the murmur of mitral regurgitation, a fact that can be shown by transplanting the insertion of the papillary muscle of dogs to another portion of the ventricular wall. The regurgitant mitral murmur will then be heard best over the new area of insertion.

A systolic murmur over the mitral or tricuspid areas occurs in the presence of insufficiency of these valves, while a systolic murmur over the pul

monic and aortic valves accompanies stenosis of these orifices Diastolic murmurs are of greater value in diagnosis When heard over the mitral area, they indicate stenosis at the aortic and pulmonic areas, insufficiency It is unwise to make a diagnosis of a valvular lesion based on the murmur alone Search should always be made for other signs that may complete the picture For example, when aortic stenosis is suspected a thrill over the aortic area accompanying the murmur strengthens the diagnosis Likewise the heart should be enlarged and show a characteristic shape on fluoroscopy and the form of the pulse wave will be suggestive When aortic regurgitation is suspected confirming evidence may be present in the peripheral circulation In making the diagnosis of valvular lesions it is also well to remember that lesions of the mitral and aortic valves are much more frequently encountered clinically while lesions of pulmonic and tricuspid valves are rare

**The murmur of patent ductus arteriosus** deserves separate comment It is continuous (both systolic and diastolic in time) and is heard with maximum intensity over the second left intercostal space The pulmonic second sound is often reduplicated is always accentuated and may even in some cases be palpable The murmur of patent ductus arteriosus is transmitted along the pulmonary artery in the direction of the clavicle

**Continuous murmurs** usually with systolic accentuation are likewise heard over other arteriovenous communications (or aneurysms) congenital or acquired A thrill may be palpable over the site of the communication which is usually in the extremities

**The peripheral circulatory signs** that point to an aortic lesion may not be present when the leak is a slight one but occur when the circulation is open and regurgitation free The increased pulse pressure that accompanies aortic regurgitation is visible in patients of medium build in a dancing or throbbing of all arteries most clearly seen in the carotids When it is present to a considerable degree in the suprasternal notch care should be taken not to make the diagnosis of aneurysm The collapsing or Corrigan type of pulse is present in aortic regurgitation the systolic blood pressure is increased and the diastolic pressure is abnormally low (usually 30 to 50 mm ) A capillary pulse may be present and abnormal sounds may be heard on auscultation over the large peripheral arteries (Duroziez s sign—pistol shot sound)

**The diastolic murmurs** most often encountered are the aortic and mitral Little trouble should arise in differentiating them if their characteristic features are kept in mind The aortic diastolic murmur has a high pitch a blowing quality and occurs in early diastole closely following the second heart sound It is usually heard best along the left sternal border when the patient is in the upright position it may be transmitted over a wide area The mitral diastolic murmur on the other hand occurs in mid or late diastole has a very low rumbling or crescendo quality and is heard best in the region of the apex beat with the patient in the left lateral position in recumbency The mitral diastolic murmur is heard over a very

small area in the region of the cardiac apex and unlike the aortic diastolic murmur is separated from the second sound of the heart by a short interval The mitral diastolic murmur is heard best with the bell type of stethoscope while the aortic diastolic murmur is more readily distinguished when the Bowles attachment is used

**Graham Steell Murmur** While aortic and mitral diastolic murmurs nearly always point to the presence of disease valve leaflets on rare occasions these murmurs may be functional For example dilatation of the aortic ring by hypertension may be accompanied by a short diastolic murmur A similar increase in the pressure in the pulmonary circuit in advanced mitral stenosis may in rare instances be associated with a functional diastolic murmur that can be heard over the pulmonic area This i the Graham Steell murmur Finally in the presence of left ventricular dilatation either secondary to hypertension or acute rheumatic myocarditis a mid diastolic murmur may be heard over the mitral area simulating mitral stenosis This diastolic murmur may disappear when the myocardium recovers from the severe infection or when the acute left ventricular strain is relieved

**Austin Flint Murmur** In some cases of aortic insufficiency the regurgitant column of blood may float the mitral leaflets into coaptation Consequently when auricular contraction occurs forcing the blood into the ventricle these leaflets vibrate and a presystolic murmur is heard in the region of the apex This is the Austin Flint murmur When present it may not be easy to differentiate from the diastolic murmur of organic mitral disease However if an apical presystolic thrill is palpable organic mitral stenosis is more apt to be the diagnosis A mall pulse instead of one of the Corrigan variety likewise suggests a mitral lesion

**For recording the signs** elicited at the different valve areas I have used the system recommended by Segall[333] This graphic method will be employed in some of the subsequent case records to avoid long descriptions Study of Fig 7 will enable the reader to become familiar with these symbols and their meanings

**Amplification Mechanisms** Until recently the unaided senses have been the only means of diagnosis of heart disease at the bedside Constant practice makes the ear of the clinician keen in its analysis of the heart sounds His skill however has natural limitations for he can only interpret those sounds that fall within the range of perception of the human ear Again the range may be variable since sounds clearly heard by one examiner may not be appreciated by another Experience and practice serve to widen the range of sound perception within certain limits beyond which man must depend on mechanical and electrical aids Both of these have been used to advantage by the research worker in extending our knowledge of cardiac sounds

During recent years rapid strides have been made following the perfection of amplification systems Electrical stetho copes have been introduced for teaching purposes and frequency filters have been improved suf

ficiently to eliminate a variety of background noises Today it is possible to pick up the heart sounds in a microphone applied over the cardiac area

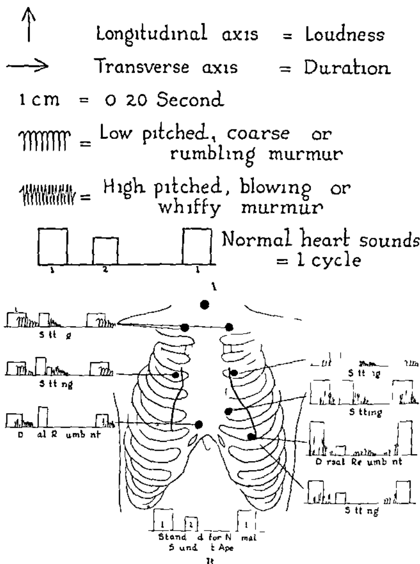



Fig 7 A Code of symbols and rules for the graphic description of cardiac sounds and murmurs Redrawn from Segall *

B The application of the graphic method Reproduction of the record of the signs recorded in a patient with aortic stenosis and insufficiency and mitral stenosis and insufficiency Redrawn from Segall *

transmit them some distance from the patient, and reproduce them on a loud speaker

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**Recording Heart Sounds** Instruments that enable the physician to record the heart sounds are now on the market The sounds of the heart are converted into electrical pulsations by a microphone of special design These pulsations amplified many times are led into the moving coil of a reflecting galvanometer and result in oscillations of a beam of light which is projected on a strip of film moving at a constant speed Part of the light beam is deflected on a ground glass scale enabling its excursions to be observed while recording Provision is also made for listening to the sounds through an electrical stethoscope (amplified auscultation)

The heart sounds may be recorded separately (stethogram) or may be recorded with the electrocardiogram (see Fig 173) If murmurs are present these will be shown by a roughening of the base line which occurs between the first and second sounds if the murmur is in systole and following the second sound if it is in diastole The patterns of these deflections may indicate the character of the murmur A murmur of high pitch for example will be recorded by many fine sharp pointed deflections while a murmur of low pitch will be represented by fewer less sharply pointed and more widely separated deflections Thrills crescendo murmurs and other sounds that may be present also appear in the graph

The results of the recording of murmurs by this apparatus have not been as satisfactory as the recording of heart sounds Many difficulties are encountered by the uninitiated that make the method one of doubtful value when used outside the laboratory under varying conditions The analysis of the records is especially difficult when vibrations other then the murmurs are encountered and recorded Considering the rapid strides made in the perfection of this instrument however the elimination of the remaining technical difficulties seems possible in the very near future

Split sounds on the other hand may be more accurately analyzed in some cases by the stethogram than by auscultation alone At times the stethogram will aid the clinician in differentiating between a third heart sound and a diastolic murmur Instead of the electrocardiogram the jugular pulse may be recorded simultaneously with the heart sounds in these records This is very useful in recognizing the difficult diastolic phases

Cautions in Amplification and Recording The physician in the field who is not in constant contact with research workers who have had experience in heart sound recording should be cautious in the purchase of this new sound equipment and in the interpretation of the records obtained Likewise if the modern practitioner tries to acquire a knowledge of the technic of all the newer laboratory methods he will find that he has little time left to maintain or increase his skill in the older methods of physical diagnosis After all the machinery of modern medicine is only an aid to our special senses and can never entirely replace them at the bedside We learn the nature of certain puzzling signs through the use of specialized instruments and if we are alert profit by this experience Consequently the more often we use them the better equipped we become to arrive at correct diagnoses unaided

Careful attention should be paid to the lungs in the examination of every cardiac patient Rales may appear at the bases in beginning congestive failure Pulmonary congestion on the other hand may occur suddenly and be accompanied by cough, frothy, blood tinged sputum dyspnea and cyanosis Numerous fine rales may be heard all over the chest, and these may quickly develop into coarse bubbling rales if fluid continues to be poured out into the pulmonary alveoli

Infarction of the lung following occlusion of the branches of the pulmonary artery by an embolus or thrombus often complicates heart disease When the infarct is small no signs or symptoms appear where a larger area is involved signs are present depending on the location and size of the vessel that is occluded If the lesion is near the outer lung margin in contact with the chest an impaired percussion note may be demonstrated and a friction rub may be heard over the same area In these cases a few localized rales can usually be detected and the patient may complain of pain at this site A cough productive of bloody sputum and an increase in the dyspnea may direct the attention of the physician to the lung fields Many times however pulmonary embolism and thrombosis are unsuspected and consequently undetected until revealed at necropsy In the course of congestive failure hydrothorax is not uncommon It is usually right sided owing to the greater likelihood of obstruction of the right azygous vein Careful examination of the back of the chest of every cardiac patient particularly those showing an increase in dyspnea, is important so that large pleural collections will not be missed

**In the examination of the abdomen** of the cardiac patient the upper right quadrant is first carefully palpated for hepatic enlargement Pain in this area may be the presenting complaint for sudden distention of the liver capsule by congestion causes marked discomfort whereas if the increase in the back pressure is gradual no symptoms may appear Pulsation of the liver accompanies tricuspid regurgitation and is rare Accumulation of fluid in the abdomen occurs in congestive failure (see Fig 2) Ascites may be present early in cases of chronic constrictive pericarditis particularly in children (page 181) The spleen is enlarged in subacute bacterial endocarditis and is a valuable finding to add to the record where a differential diagnosis is sought between this disease and a recurrence of rheumatic infection Abdominal pulsations are common especially in women but the diagnosis of abdominal aortic aneurysm requires the palpation of a definite mass showing expansile pulsation (page 485)

**Examination of the extremities** will furnish valuable data Pulse differences should always be searched for Clubbing of the fingers and toes may occur in subacute bacterial endocarditis When associated with cyanosis clubbing usually points to a diagnosis of congenital heart disease (see Fig 124) The elbows ankles, wrists and knees should be carefully inspected for evidence of rheumatic nodules (see Fig 54) The appearance of these small subcutaneous nodes often aids in the diagnosis of the rheumatic state All reflexes should be tested Signs pointing to a syphilitic

infection of the nervous system may throw light on the nature of an aortic lesion when the etiology is in doubt The legs should be carefully inspected for the presence of edema

## ROENTGEN METHODS IN DIAGNOSIS

While a careful history and physical examination followed by the correct evaluation of all symptoms and signs elicited should enable the physician to manage properly and efficiently the majority of cardiac patients whenever possible at the end of the clinical examination when diagnostic impressions have been recorded a roentgen study should be made[370] This extends the power of inspection a little deeper and comprises the last part of the physical examination of the cardiac patient It may only confirm the diagnosis However in doubtful cases especially where the difficulties of physical examination have been increased by such conditions as obesity emphysema or pregnancy, the roentgen examination furnishes additional information of great value

Before any drawings of the heart outline (orthodiagrams) or roentgen films of the chest (roentgenograms) are made a great deal of information concerning the heart and great vessels can be obtained by fluoroscopy The size and shape of the heart can be seen at a glance By turning the patient in various positions all the cardiac chambers may be studied and their pulsations noted The pulmonary fields are open to inspection and engorgement can be detected while the size density and course of the aorta can be determined Today all of this data is added to the patient s record by the clinician who has invaded the field of the roentgenologist to the extent of making his own fluoroscopic examinations and orthodiagrams It is therefore possible for the entire cardiac study to be completed before the patient leaves the office

Some clinicians still prefer to take roentgenograms with the patient in various positions and to study these later for abnormalities of cardiac size and shape To be satisfactory for this purpose roentgen films must be taken with the tube at a distance of at least seven feet from the patient's chest. In this way only parallel rays from the tube are used and distortion of the cardiac silhouette is avoided (Fig 8) The nearer the tube is moved to the patient the greater will be the distortion of the cardiac shadow At a distance of seven feet or more the rays from the tube are practically parallel but an exact reproduction of the heart size on the film is not obtained The percentage of error however is negligible and the method is suited for clinical purposes[317]

The orthodiagram on the other hand has many advantages To begin with it is much less expensive The equipment necessary consists of a fluoroscope properly fitted with a few special attachments for making orthodiagrams (Fig 9) and the supplies (parchment tracing paper planimeter copying pencils and slide rule) for making and calculating the orthodiagrams[197] True cardiac size is obtained for here the tube is moved by

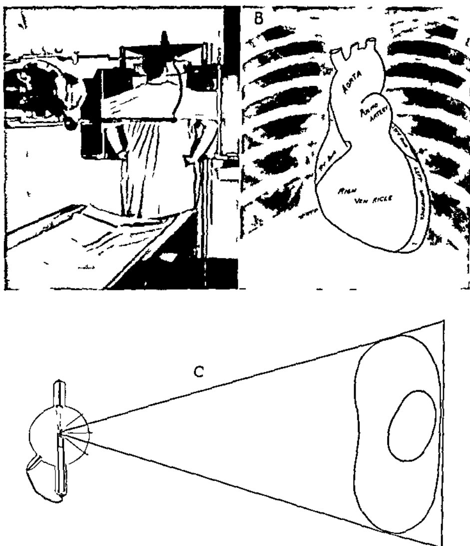



FIG 8 The teleroentgenographic method

A The patient in position with the tube at a distance of seven feet

B The roentgen film The various cardiac chambers are indicated on the silhouette

C Diagram illustrating course of divergent rays It can be seen that the smaller the distance between the tube and the patient the greater will be the distortion on the screen

Advantages of method 1—A standard procedure that can be carried out by a technician 2—This result is more accurate if the orthodiagraphic measurements are made by an inexperienced observer

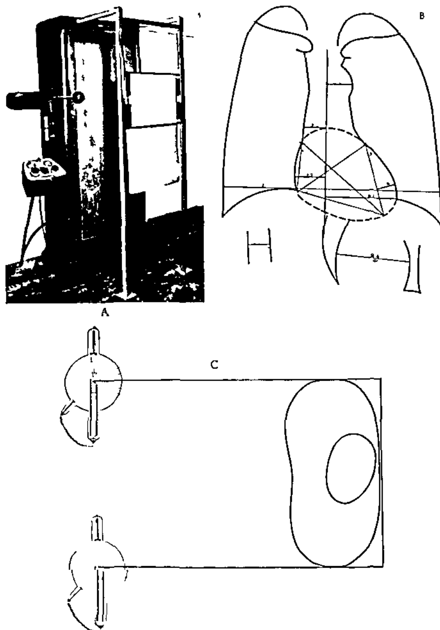



Fig 9 The orthodiagraphic method A Fluoroscope with orthodiagraphic attachment (Courtesy Westinghouse X Ray Corporation) B The orthodiagram C Diagram illustrating the principle of projection Parallel rays are obtained by moving the tube and no distortion of the image results

the operator and a small beam of parallel rays, controlled by a shutter, is used to illuminate the cardiac border Experience reduces the incidence of technical errors Finally, it is desirable that the clinician who makes the physical examination should also carry out the roentgen study In this way he can check his findings at once and in addition to determining accurately cardiac size he sees the heart contour in the various positions and can closely study cardiac contractions Valuable data not obtainable at the initial examination may be furnished by a series of studies made on the same patient by the same observer at intervals (see Fig 24)

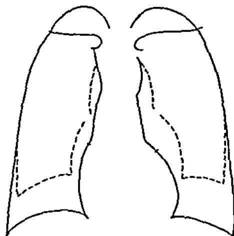


FIG 10 Diagram illustrating the effect of respiration on cardiac position The solid line represents inspiration The broken line represents the position of the heart in expiration

A number of factors influence the size and shape of the normal cardiac silhouette observed during fluoroscopic examination If the respiratory movements are forced distortion of the shadow will result With deep inspiration the heart becomes long and narrow with deep expiration short and broad (Fig 10) The patient should therefore be instructed to breathe normally and slowly In recumbency increase in the return volume of blood entering the heart will slightly widen the shadow, while in the erect position a decrease in the return flow is apt to give a slightly smaller silhouette If the standing position is used all subsequent observations made for purposes of comparison should be carried out in this position

Fluoroscopy may be increased in value if the patient swallows barium paste during the examination This outlines the esophagus and enables its relationship to heart and aorta to be determined (Fig 11)

In every fluoroscopic examination of the heart the patient should be studied in four standard positions

**Anteroposterior Position** In this view the heart appears as a dense shadow in the middle of the chest with the apex directed toward the left.




A Displacement of the esophagus by enlargement of the left auricle Patient in the right oblique position




B Kreuzfuchs method for determination of the aortic diameter Patient in anteroposterior position (See text)




C Right sided aorta Patient in the right oblique position (Courtesy of Dr E P Pendergrass)

Fig 11 The value of the barium filled esophagus in roentgen examinations of the heart and great vessels

Wide variations in cardiac shape may be seen to occur normally, and these will depend upon the body build For example if the diaphragm is high as in individuals of hypersthenic habitus the heart will be elevated and will appear wider (Fig 12A) while in the hyposthenic type the diaphragm is low and the heart assumes a central vertical position (Fig 12B)

In the anteroposterior position the upper curve of the right border (Fig 13) follows closely the right sternal margin and is formed by the great vessels in younger patients the superior vena cava usually occupies

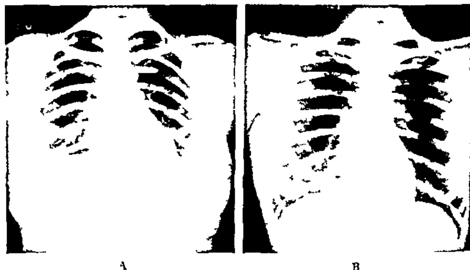




Fig 1 Influence of bodily conformation on the cardiac position and shape
A The hypersthenic type The diaphragm is high
B Hyposthenic type The diaphragm is low (ptotic or drop heart)

this position but in older patients the aorta is seen In the latter instance an arterial type of pulsation is noted in the convex shadow of this region The lower half of the right border curving outward and downward to the cardiophrenic angle is formed by the right auricle Rarely the inferior vena cava and cardiodiaphragmatic ligament become visible below the auricle

Viewing the cardiac silhouette in the anteroposterior position from above downward on the left side the first structure that attracts attention is the aortic knob forming a short curve convex to the left In older patients this may be quite prominent The next small arc seen on the left border varies in its prominence in different patients and is formed by the pulmonary artery It exhibits a lesser degree of arterial pulsation than is usually seen in the aortic area The pulmonary area may be quite convex and prominent in children and young adults but this decreases with advancing years A small portion of the left auricular appendage can usually be seen on the left border inferior to the pulmonary artery Enlargement or

bulging in this region has considerable significance in rheumatic heart disease when mitral stenosis and left auricular enlargement are present The left ventricle constitutes the remainder of the left border amounting to nearly one half of the total length It is usually slightly convex outward and

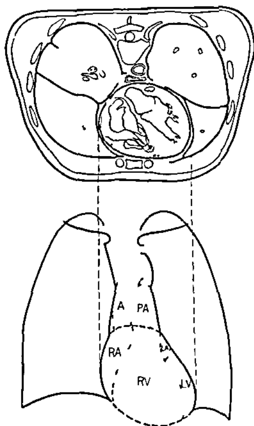



Fig 13

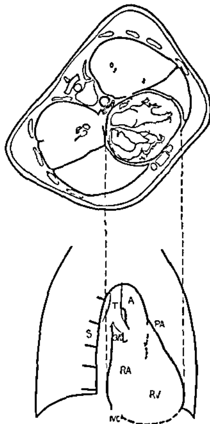


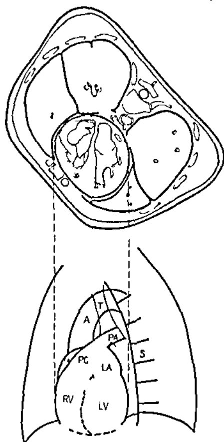

Fig 14

Fig 13 Schematic diagram illustrating topography of the heart and great vessels with the patient in the anteroposterior position

Fig 14 The topography of the heart with the patient in the right oblique position

curves sharply around the apex of the heart At times an air bubble in the stomach may permit visualization of the inferior cardiac border (see Fig 103) but nearly always this part of the outline is lost in the subdiaphragmatic shadows The inferior cardiac border is composed of the left ventricle for a small distance in the region of the apex The right ventricle makes up nearly one half of the remaining distance to the sternum while the right auricle completes the inferior border

**Right Anterior Oblique Position** The examination is continued with the patient in the right anterior oblique position (sometimes called the first oblique or right oblique position) Here the right shoulder remains in touch with the screen while the body is rotated through an angle of approxi



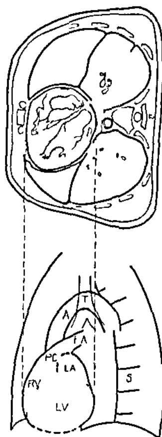




Fig 15 Fig 16

Fig 15 Diagram illustrating the topography of the heart and great vessels with the patient in the left anterior oblique position

Fig 16 Diagram illustrating the topography of the heart and great vessels with the patient in the lateral position

mately 40 degrees (Fig 14) Standard degrees of rotation are unsatisfactory It is best to turn the patient slowly until the cardiac silhouette, as shown in the figure is seen to its fullest extent The right or posterior border of the shadow is now composed of the superior vena cava the right auricle and the inferior vena cava The anterior border is made up of the ascending aorta the pulmonary artery and the right ventricle The right anterior oblique position is most important where early mitral stenosis is suspected The cardiac silhouette in this position will then be altered by the encroachment of the left auricle on the retrocardiac space (see Fig 11)

**Left Anterior Oblique Position** In the left anterior oblique or the second oblique position the patient's left shoulder remains in contact with the screen and he is rotated to the right about 40 degrees This position is particularly suitable for visualization of the aorta The ascending portion can be clearly seen the transverse portion less distinctly, while the descending portion may be followed until it merges with the vertebral column shadow The anterior border of the heart in this position (Fig 15) is formed by the aorta the right auricle and the right ventricle The trachea may be seen at its bifurcation between the ascending aorta and the spinal column This air-containing structure tends to obscure the transverse aortic arch It is important to note that in the left anterior oblique position all the chambers of the heart can be visualized

**Lateral Position** For this view the patient stands at right angles to the screen with the left shoulder forward and both hands behind the head Here the anterior border of the heart (Fig 16) is made up of the aorta and the right ventricle while posteriorly beginning below the arch the border is made up of the pulmonary artery the left auricle the left ventricle and at times a portion of the inferior vena cava The area between the back of the heart and the vertebral column again visualized clearly in this position is the retrocardiac space It is usually clear and normally averages about 2 to 3 cm in width unless the anteroposterior diameter of the chest is small in which event it may be proportionately narrowed or entirely obliterated A careful study of the retrocardiac space with the patient in the oblique position is most essential since both left atrium and left ventricle are posterior chambers and their enlargement may be detected by encroachments upon this area

## Cardiac Size

Cardiac hypertrophy except in a few rare instances means heart disease consequently it is of primary importance to determine the size of the heart While percussion may furnish this evidence roentgen studies are valuable in checking the result and in pointing out the degree of participation of the various cardiac chambers in the enlargement A number of methods for measuring and recording the heart size are in use Of course the first impression as to the presence or absence of cardiac enlargement is gained at the time of the fluoroscopic examination The relationship between the size of the heart and the diameter of the chest is first noted Later this cardiothoracic ratio may be calculated from the roentgen film or orthodiagram To obtain this figure from the orthodiagram drop a perpendicular line of the chest starting midway between the sternoclavicular joints (Fig 17) At right angles to this line draw the transverse diameter of the chest tangent to the dome of the right diaphragm The transverse diameter of the heart is obtained by adding RM (greatest extension of the heart to the right) and LM (greatest extension of the heart to the left) If this figure is divided by the transverse diameter of the chest the result will be the cardiothoracic ratio The normal value is 0 5 but values in

excess of this are not in themselves indicative of cardiac enlargement Alterations in the heart position (Fig 10) and differences in body build (Fig 12) will cause the cardiothoracic ratio to show wide variations

The most valuable criterion in estimating cardiac enlargement is the cardiac area of the orthodiagram In measuring this surface the antero-posterior view must be completed by drawing arbitrary lines to represent

FIG 17 A normal orthodiagram (For explanation see text)

the upper and lower cardiac borders It is then possible to determine the number of square centimeters in the area by the use of a planimeter (Fig 18A) The result can be compared to the predicted value obtained from tables of Hodges and Eyster and the percentage of deviation from the normal noted Early cardiac enlargement may be detected in this manner Normal transverse diameters based on the age height and weight have likewise been estimated and should be included in all cardiac studies A cardiac slide rule made up from the Hodges Eyster formulae (Fig 18B) is available and is useful in quickly obtaining the predicted cardiac area and transverse cardiac diameter of any adult patient

Various diagonals to connect points on the orthodiagram have been

recommended by different workers as a guide to the enlargement of the individual chambers of the heart It is necessary to place certain markings on the cardiac border during the fluoroscopic examination before these diagonals can be drawn (Fig 17) The point (A) on the right cardiac border designates the junction of the superior vena cava and the right auricle (B) the right cardiodiaphragmatic angle (C) on the left border marks the junction of the auricular appendage and the ventricle Incidentally point (C) is not always easy to locate It requires close inspection along the left border during the fluoroscopic study to discern the dividing point or fulcrum where the type of pulsations changes

When these points are marked the orthodiagram is traced from the screen on parchment paper and placed on a drawing board B and C are connected A ruler is placed parallel to the line BC and tangent to the lower end of the cardiac border The point where the border touches the ruler is marked D This approximates the cardiac apex The following lines are now drawn AB this is the chord of the right auricle AD this is the long diameter of the heart A perpendicular line from PP to C(SC) represents the left auricle Other perpendicular lines are drawn from the midline of the chest to the most distant points on each cardiac border (RM and LM) Next connect the points C and D This distance is known as the chord of the left ventricle Now connect B and D This is the chord of the right ventricle A perpendicular from the line CD to the outermost part of the left ventricle is now drawn and marked N This represents the rise of the left ventricle A similar perpendicular from AB to the right auricular border is drawn and marked O This represents the rise of the right auricle To measure the width of the great vessels in the supracardiac shadow perpendicular lines are drawn from the midline of the chest to points of farthest extension to the right (1) and to the left (2) The sum of these should not exceed 7 5 cm but the value will vary depending on age build and amount of tortuosity of the aorta

The aorta may usually be measured in the right and left anterior oblique positions at which time the diameter may be marked on the screen with the patient rotated to the angle giving the greatest visibility and using the smallest possible pencil of parallel rays from the tube In the anteropo terior position the aortic diameter at the beginning of the descending portion may be obtained by using the procedure recommended by Kreuzfuchs This method is based on the anatomic fact that the right side of the descending aorta in the region of the aortic knob parallels the left side of the esophagus As the patient swallows barium paste the left side of the barium column and the left border of the aorta are marked on the screen (Fig 11) From this measurement 3 mm, representing the width of the esophageal wall is deducted

## Characteristic Orthodiagraphic Changes

Certain types of heart disease especially those associated with valvular lesions alter the orthodiagram in a characteristic manner In mitral regurgitation there may occasionally be noted an outward pulsation in the region

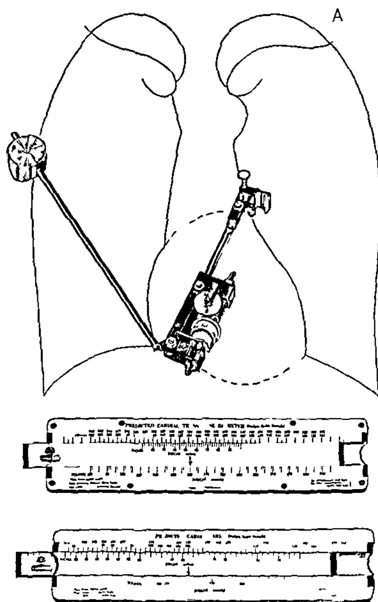



FIG 18 A Calculation of the cardiac area by the use of the planimeter B The cardiac slide rule *

* Manufactured by the Picker X Ray Corporation 300 Fourth Ave N Y

of the left auricle on the left cardiac border occurring during cardiac systole The orthodiagram will show enlargement to the left and downward of the left ventricle (Fig 19)

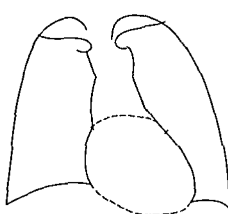


Fig 19 Mitral regurgitation

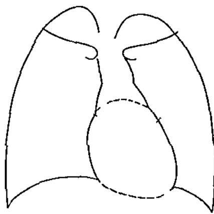


Fig 20 Mitral stenosis

**Mitralization** In mitral stenosis the cardiac silhouette shows characteristic alterations The thin walled left auricle responding to the strain enlarges and the region occupied by this structure on the left cardiac border becomes more conspicuous At the same time increased pressure in the pul

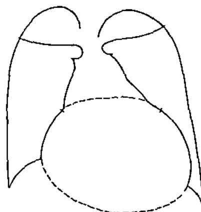


Fig 21 Advanced mitral stenosis and regurgitation

monary circuit causes bulging of the region occupied by the pulmonary artery just above the auricular segment The result (Fig 20) is a straightening of the whole left border of the heart This is spoken of as mitralization The effect is further accentuated by a decrease in the size of the aortic knob

Progress in the mitral lesion tends to reduce the amount of blood entering the left ventricle consequently this chamber does not increase in size—



Fig Advanced mitral stenosis Note the bulge on the right cardiac border caused by the dilatation of the left auricle

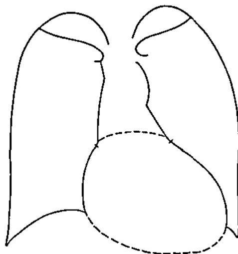
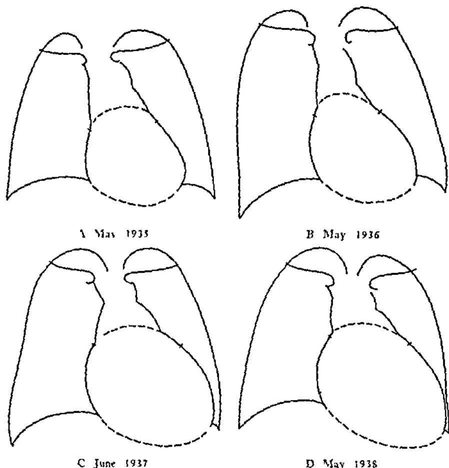


Fig 23 Aortic regurgitation

in some cases even becomes smaller Since both left auricle and left ventricle occupy posterior positions early changes may be detected by turning the patient and noting encroachments on the retrocardiac space in either

the right oblique or lateral views (Fig 11) The enlargement of the left auricle in mitral stenosis may be seen in the upper and middle thirds of the retrocardiac space When there is an accompanying mitral regurgitation with left ventricular enlargement the lower third of the retrocardiac space is likewise narrowed by the increase in size of the left ventricle In



A May 1935 B May 1936

C June 1937 D May 1938

Fig 4 Serial orthodiagrams from a case of rheumatic heart disease with regurgitation and stenosis at mitral and aortic valves Note progressive increase in cardiac size over a period of four years

advanced cases with generalized cardiac hypertrophy the entire retrocardiac space may be obliterated

A greater degree of mitral stenosis and regurgitation causes the heart to assume a triangular shape (Fig 21) This is caused by the combined effect of the two lesions The whole of the left border shows mitralization but in addition there is enlargement of the heart to both right and left

At times in advanced mitral stenosis the left auricle may become so large that it may be seen extending beyond the right cardiac border (Fig 22)

Aortic regurgitation produces left ventricular enlargement (Fig 23) Other conditions such as aortic stenosis and hypertension place a strain on the left ventricle and are accompanied by similar alterations in the cardiac silhouette The aortic knob becomes more prominent and the pulsations in aortic regurgitation will be seen to have an increased excursion In addition to these changes, in patients with syphilitic aortitis and aortic regurgitation the aorta may show considerable increase in density If the



Fig 5 Large pericardial effusion

patient is viewed in the left lateral position the increased size of the ventricle will be indicated by the degree of encroachment on the retrocardiac space In this position the left ventricle occupies the lower two-thirds of the posterior border consequently slight variations in its size may be detected

Combined aortic and mitral lesions alter the cardiac silhouette in proportion to their relative severity Usually all chambers are enlarged (Fig 24) The left ventricular size is always increased and rotation of the patient to the right oblique position shows a marked degree of obliteration of the retrocardiac space by the dilated left auricle

Lesions of the tricuspid valve are rare The clinical diagnosis of tricuspid regurgitation is difficult but fluoroscopically it may be suspected when an unusual degree of pulsation coinciding with ventricular systole is noted along the right auricular border accompanied by pulsation of the superior vena cava Stenosis of the tricuspid valve another rare lesion, will

produce right auricular enlargement with some dilatation in the region of the superior vena cava

Large effusions into the pericardial sac produce a typical alteration of the configuration (Fig 25) This is known as the leather water bottle shape Smaller effusions may be seen in the dependent position of the sac with the patient in the upright position Alterations should be searched for just above the diaphragm where the angle between the liver and the peri cardium may become acute In pericarditis with effusion close inspection of the borders of the cardiac silhouette will show decreased pulsations As the fluid accumulates further the pulsations may entirely disappear and the usual cardiac outline may be obliterated Care should be taken not to



A B

Fig 26 Calcium deposits in the aortic valve (Marked by arrow) This patient was a male of 56 who gave a history of several attacks of acute rheumatic infection in childhood A Right anterior oblique position B Left anterior oblique position

mistake the dilated heart with feeble pulsations for this picture of peri cardial effusion When in doubt roentgen films taken both in the upright and recumbent positions may be helpful In recumbency it will be noted that the heart becomes globular in shape owing to a more even distribution of the fluid contained within the pericardial sac

Chronic pericarditis should be suspected when the cardiac silhouette is smaller than one would expect to encounter considering the severity of the clinical signs (see Fig 82) In this type of heart a diminution in the pulsa tion is likewise a characteristic feature

Roentgen films particularly the lateral and oblique views may show calcium deposits and these will be of great assistance in the diagnosis

(Fig 26) Fixation may be demonstrated in chronic mediastinopericarditis by taking roentgenograms in various positions and noting the shift of the cardiac border

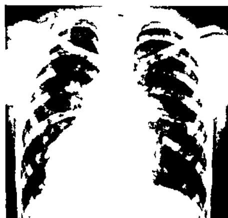




Fig 27 Fig 28

Fig 27 Congenital dextrocardia This patient was a child of 10 who presented in addition the typical signs of an established mitral stenosis Note the cardiac enlargement and mitralization

Fig 28 Patent ductus arteriosus

Congenital cardiac defects occur in a variety of combinations A careful analysis of the contour of the orthodiagram and a consideration of the findings on physical examination will often enable the physician to diagnose correctly the type of congenital abnormality present True congenital dex

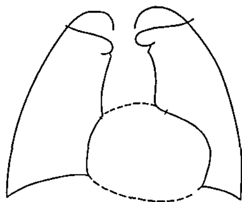


Fig 29 Pulmonary stenosis (Coeur en sabot)

trocardia (Fig 27) presents no difficulty Patent ductus arteriosus in some cases may cause no alteration in the cardiac silhouette If the lumen of the communication is large cardiac size will be increased (Fig 28) and a

prominence will be noted in the region of the pulmonary artery with increased pulsations over this area In stenosis of the pulmonary valve increase in the size of the right ventricle is common This tends to elevate the apex of the left ventricle, producing a cardiac configuration resembling

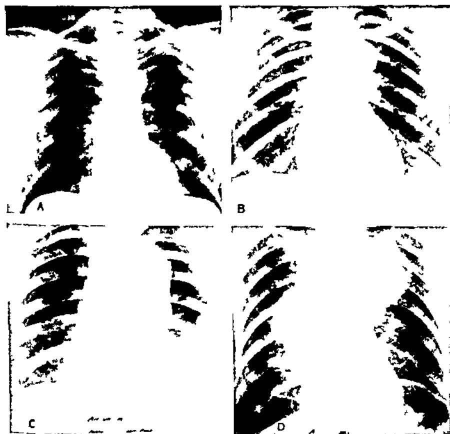



FIG 30 Syph litic cardiovas ular disease
A Early syph lit c aortitis Note th enlargement of the ascend ng aorta
B Dilated ascending a rta (early aneurysm)
C Aneurysm of the entire aortic arch
D Saccul r an urysms

a wooden shoe the so called coeur en sabot (Figs 29 and 136) In coarctation of the aorta the ascending aorta may be dilated and increased pulsations will be observed The congenital band constricting the aorta increases the work of the left ventricle and hypertrophy of this chamber may result The intercostal arteries serving as collateral pathways in these cases may erode the lower borders of the ribs This change can

usually be demonstrated only by means of a satisfactory roentgen film (see Fig 128)

Measurements of the aorta are occasionally of importance to the clinician in diagnosis In syphilitic aortitis the early changes can be detected only by roentgen examination Experience in the fluoroscopic method increases the ability of the clinician to recognize these differences in aortic density In syphilitic aortitis the aorta may appear on the right cardiac border or the aortic knob may be prominent and the changes in the aortic wall may produce a wider range of pulsations Portions of the aortic wall may weaken early in the process and local bulgings may be seen fluoroscopically long before they can be recognized clinically The aorta should be studied care

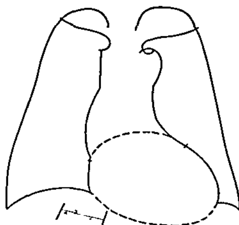


FIG 31 Orthodiagram of a patient suffering from syphilitic cardiovascular disease Aortic regurgitation was present Note aortic dilatation and cardiac enlargement The aortic measurement recorded below was made with the patient in the right anterior *oblique position*

fully in all positions in suspected cases The most common site for aortic aneurysm is in the ascending arch near the base of the heart (Figs 30 and 31) These aneurysms are usually associated with aortic regurgitation and left ventricular enlargement Where the aortic valves are not affected by the syphilitic process and where the mouths of the coronary arteries escape, even large aneurysms may not cause any change in the cardiac size or shape Aneurysms of the transverse arch of the aorta (Fig 32C) by pressure on neighboring structures may cause cough hoarseness or dysphagia Indentation or displacement of the barium filled esophagus by the dilated transverse arch may be readily demonstrated Serial orthodiagrams are useful in following changes in the aorta during treatment

In hypertension the aorta becomes prominent assumes a position on the right cardiac border where increased pulsations can be noted synchronous with ventricular systole At the same time cardiac hypertrophy

Fig 32 Syphilitic cardiovascular disease
A Aneurysm of ascending aorta and innominate
B Fusiform aneurysm descending aorta
C Aneurysm ascending and transverse arches
D Same as C four months later A fatal hemorrhage took place into pleural cavity

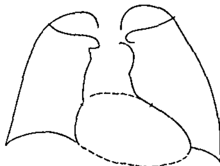


Fig 33 Hypertensive cardiovascular disease

is seen (Fig 33) In the presence of advancing sclerosis of the aorta areas of density corresponding to calcified areas may be detected (Fig 34)

It must be kept in mind at all times that serious heart disease may be present with no alterations in the size or shape of the cardiac silhouette This is true in some cases where coronary sclerosis exists as the single lesion although following occlusion enlargement is quite apt to develop In fluoroscoping all cases that give a history of previous coronary occlusion a close watch should be kept for unusual bulgings in the cardiac outline suggestive of cardiac aneurysms (Figs 35 and 110)

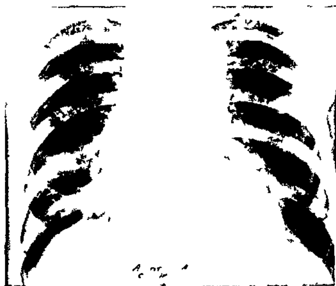


Fig 34 Arteriosclerotic aortitis with calcification

Study of the heart action as a whole during fluoroscopy gives valuable information Auricular contractions precede the ventricular by 0 10 to 0 20 second but this difference can seldom be appreciated by inspection of the silhouette However in cases of heart block auricular contractions may be clearly seen to precede the ventricular contractions The aorta and pulmonary artery are prominent during cardiac systole and stand out dis*tinctly on the left cardiac border Exaggeration of this movement* is seen in some pathologic conditions such as aortic regurgitation and hypertension The whole heart may be seen to have increased excursions in patients suffering from hyperthyroidism However it is important for the beginner to remember that THE CHARACTER AND THE EXTENT OF THE CARDIAC PULSATIONS OBSERVED FLUOROSCOPICALLY SHOULD NEVER BE TAKEN AS AN INDEX OF CARDIAC FUNCTION Weak contractions are encountered in patients suffering from myocardial insufficiency but may also be seen in normal patients who are obese and have high diaphragms with so called transverse hearts

In addition to the roentgen film or teleoroentgenogram and the ortho

diagram another method of study has recently been perfected By means of a grid that moves at a definite rate as exposures are made shadows are obtained that reveal the maximum and *minimum excursions of the cardiac* borders This is known as a kymogram It requires specialized technical

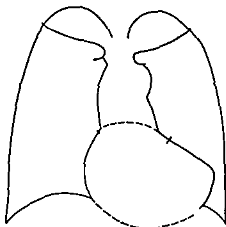


FIG 35 Cardiac aneurysm

methods and is generally employed by those possessing special roentgeno logic training

## ELECTROCARDIOGRAPHY

The electrocardiograph is an outstanding example of the usefulness of the laboratory in clinical medicine Invented and perfected by the com bined efforts of many laboratory workers over the course of half a century it has proved worthy of the high place it holds in medical practice and every physi ian should possess a knowledge of at least the fundamental principles of the method

The electrocardiogram should always be interpreted in the light of the clinical findings The physical methods of diagnosis properly applied usually yield more abundant evidence than the electrocardiograph for just as the physical examination may be negative in some cases of advanced coronary disease or the neurologic signs produced by some brain tumors insignificant so the electrocardiogram may be negative in the presence of such serious conditions as angina pectoris or subacute bacterial endo carditis The graphic method of electrocardiography after all only shows the origin and distribution of the impulse for cardiac contraction It locates accurately an abnormal origin of the impulse and detects the site of any disease process that interferes with its spread but it gives no clue as to the nature of the disease

**Arrhythmias** A knowledge of the cardiac arrhythmias can be very

quickly gained by a study of the alterations they produce in the electrocardiogram In fact, introducing a greater accuracy of diagnosis into the field of the irregularities was an initial task successfully and quickly completed by those working with the electrocardiograph The physician was soon able through the knowledge gained from the study of the electrocardiogram to diagnose unaided nearly all the cardiac irregularities In some instances, the instrumental method must still be called upon to render the final opinion For example if a sinus arrhythmia, a benign condition in itself is accompanied by frequently recurring premature beats or extra systoles the differentiation from auricular fibrillation may be difficult If premature beats alone are observed and diagnosed clinically, it is still desirable to have an electrocardiogram then their point or points of origin may be determined with accuracy If they arise from more than one focus this fact will be shown by the tracing in which event the extra systoles are more apt to accompany organic myocardial disease Furthermore any additional changes that may be encountered in the electrocardiogram may shed more light on the diagnosis

**Infection** When cardiac infection is suspected the electrocardiogram is invaluable In some cases it will furnish the only positive evidence upon which a diagnosis of acute myocarditis can be based For example if an inflammatory process involves the conduction system of the heart delay in the transmission of the contraction impulse may occur At times the impulse may be completely blocked but usually the changes produced in the tracing are slight and transient If these are detected in the absence of cardiac enlargement or other decisive clinical signs the electrocardiogram becomes a valuable aid in establishing the diagnosis and instituting proper therapy

**Paroxysmal rapid heart rates** are often puzzling Although the physician unaided can usually make the correct diagnosis at the bedside the electrocardiogram can be a great help at times For example in paroxysmal tachycardia the electrocardiogram shows whether the paroxysm is of auricular or ventricular origin The ventricular variety is much more serious from the standpoint of prognosis and is more apt to be associated with heart disease The early recognition of the presence of ventricular tachycardia may serve as a warning for it often precedes the onset of ventricular fibrillation The electrocardiogram is also useful in the diagnosis of auricular flutter particularly where high degrees of block are present making an accurate clinical diagnosis difficult

**Essential Hypertension** In the course of essential hypertension evidence of cardiac changes should always be sought in the electrocardiogram If myocardial damage is suggested the regime of treatment should be altered accordingly to lessen the strain on the cardiac reserve

**In angina pectoris** a positive electrocardiogram may be valuable when this diagnosis is in doubt although many times the tracing in angina is negative Electrocardiograms taken during an attack of anginal pain

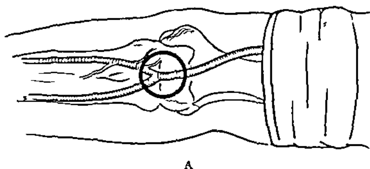
following exercise, or induced anoxemia may show slight alterations in the form of a graph that was previously normal and furnish the necessary objective evidence

**Drug Effects** Drugs alter the appearance of the electrocardiogram As we would expect digitalis produces the most marked and characteristic change and the action of increasing doses is reflected in the rate, rhythm and form of the individual waves Consequently the electrocardiogram is very valuable in giving warning of approaching toxic symptoms that not infrequently occur during the administration of this drug When quinidine is indicated in an attempt to restore normal rhythm in cases of auricular fibrillation and flutter its action can be carefully watched by frequently repeated electrocardiograms

**Valvular Lesions** Occasionally in puzzling cases where valvular lesions are present the electrocardiogram may act as a final court of appeal For example when pulmonary stenosis is suspected the diagnosis is much more likely to be correct if the tracing shows a right axis deviation Aortic stenosis is characterized by a left axis deviation The auricular changes that accompany mitral stenosis often place their mark on the P waves of the electrocardiogram It is readily seen that these various signs may become useful when the clinical signs are inconclusive

**Myocardial Disease** While the earlier field of usefulness of the electro cardiograph was the analysis and classification of the various arrhythmias in recent years the detection of myocardial disease has been its most important function In no other condition is the electrocardiogram as valuable as in coronary thrombosis With the use of the new chest leads the diagnosis of acute myocardial infarction can be made with certainty in over 90 per cent of the cases Occlusion of one of the main coronary arteries or any of its branches produces characteristic changes in the form of the electrocardiogram many times when the remainder of the cardiac examination is entirely negative The alterations that appear are transient and the electrocardiographic picture may change from day to day consequently frequent tracings are necessary in guiding the therapy in any case In spite of the great service rendered by the electrocardiograph in this condi tion it must be stated that the very small percentage of the cases giving the typical clinical history of coronary thrombosis in which the tracing is negative should always be treated on the basis of the clinical findings

The conclusion can be drawn that the electrocardiographic tracing is a valuable supplement to the examination of every cardiac patient It is an aid in the diagnosis of the arrhythmias a guide to the administration of drugs and reaches its peak of usefulness often when other methods of diagnosis fail in the detection of coronary thrombosis A full discussion of the fundamental principles of electrocardiography and a detailed study of the normal and abnormal electrocardiograms for the reader who is not familiar with the form of these curves will be found in Chapter 24



A

B

FIG 36 Estimation of the arterial blood pressure by the auscultatory method
A Position of the stethoscope in relation to the brachial artery
B The apparatus and technic (Courtesy of the Taylor Instrument Company)

## SPHYGMOMANOMETRY

Most of the models of blood pressure instruments now on the market are satisfactory and are becoming sufficiently rugged in construction to stand the strain of long usage (Fig 36B) Errors under five mm in clinical models that receive ordinary care should not cause alarm More often wide differences in readings are due to the technic used by the observers Recently the committee for the standardization of methods of taking blood pressure readings appointed by the American Heart Association and by the Cardiac Society of Great Britain and Ireland have published suggestions in an attempt to standardize the method of taking and recording the blood pressure readings[3 1] These suggestions summarized below should be given careful consideration by every practicing physician *

1 The equipment should be kept in good condition and calibrated at yearly intervals

The patient should be comfortably seated with the arm free of constriction and supported at heart level on a smooth surface If the readings are taken in any other position notation should be made

3 A standard cuff with rubber bag 1 to 13 cm in width should be used The rubber bag should be applied to the inner aspect of the arm one inch above the ante cubital space Neither bulging nor displacement of the cuff should occur on inflation

4 Palpation should always be used to check results The pressure in the cuff should be quickly increased in steps of 10 mm of mercury until the radial pulse ceases, and then allowed to fall rapidly If the radial pulse is felt at a higher level than that at which the auscultatory sound is heard the palpatory reading should be accepted as the systolic pressure otherwise the auscultatory reading should be accepted

5 In making auscultatory readings the stethoscope should be placed over the previously palpated brachial artery in the ante cubital space (Fig 36A) not in contact with the cuff No opening should exist between the lip of the stethoscope and the skin this should be accomplished with the minimum of pressure possible The hand may be pronated or supinated according to the position yielding the clearest brachial pulse sounds

6 To obtain systolic pressure inflate to a pressure about 30 mm above the level at which the radial pulse can be palpated The cuff should then be deflated at a rate of from two to three mm of mercury per second The level at which the first sound regularly appears should be considered the systolic pressure unless as already described the palpatory level is higher in which event the palpatory level should be accepted This should be noted

7 With continued deflation of the cuff the point at which the sounds suddenly become dull and muffled should be known as the diastolic pressure If there is a difference between that point and the level at which the sounds completely disappear the American Committee recommends that the latter reading should be regarded also as the diastolic pressure This should be recorded in the following form RT (or LT) 140/80 70 or 140/70 0 If these two levels are identical the blood pressure should be recorded as follows 140/70 70 The cuff should be completely deflated before a further determination is made The British Committee believes that except in aortic regurgitation it is nearly always possible to decide the point at which the change occurs and this is the only reading that should be recorded

These specific recommendations should be carefully followed Certain minor points likewise deserve consideration Either mercurial or aneroid instruments are capable of

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correct readings if kept in good condition To this end the mercury manometer should be checked for

(1) Level of mercury It should be kept at zero mark

(2) Small air vent at top of glass tube should be kept clear Incorrect readings occur if this is neglected

(3) Instrument should be kept on level surface and on level with observer s eye for correct readings

A yearly calibration for aneroid type of instrument is recommended since a fall or a blow causes inaccuracies due to changes in aneroid diaphragm The needle should stand at zero when the apparatus is completely deflated and should move immediately when inflation begins Manometers which have a stop pin at zero or those with rotatable dial permitting the user to set the zero mark anywhere are not recommended

All valves should be frequently inspected and should function smoothly The entire system should be free of leakage Rubber cuffs should be 12 to 13 cm wide and 23 cm long The cloth covering should be inextensible material so that even pressure is exerted throughout the width of the cuff it should extend as a band 15 cm wide for 60 cm *beyond the edge of the rubber cuff and then taper gradually during the additional* 30 cm The new style of cuff using slide fastener device is acceptable If bulging occurs above and below the band the reading may be inaccurate

*A special cuff should be used to record blood pressure of the leg* The rubber bag should be 15 cm wide and 30 cm longer than in the case of the armlet (total 120 cm ) For children cuffs of the following widths have been suggested under eight years less than 9 cm under four years less than 6 cm new born less than 2 5 cm The limited work done in this field does not warrant a definite recommendation at this time For blood pressure readings of the thighs the auscultation should be over the popliteal artery with the patient prone

**Physical and Physiologic Factors** Activity alters the blood pressure consequently the patient should rest for a short period if possible before a reading is made Emotion at the time may likewise influence the result therefore the first reading is nearly always higher Later readings on subsequent visits will show the true blood pressure level

The blood pressure varies at different times of the day In important cases the time of the day should be noted and subsequent observations should be made at essentially the same time in the same relationship to meals sleep exercise and other factors If the physician wishes to minimize the sources of error several blood pressure readings should be made and the highest and lowest pressures of the series recorded

The determination of blood pressure in arrhythmias is unsatisfactory When premature contractions are present the higher systolic pressure of the beats that terminate compensatory pauses should be ignored With auricular fibrillation both systolic and diastolic readings should be recorded as being approximate only Alternation of the pulse (pulsus alternans) during blood pressure determinations may indicate left ventricular weakness

When especially careful studies of the blood pressure are to be made the use of basal blood pressure conditions should be considered The preparation should be similar to that used in making basal metabolism tests The basal blood pressure determination should be made from 10 to 12 hours after a previous meal (preferably in the morning) after the patient has rested 30 minutes in a comfortably warm room and is men

tally as well as physically at ease This procedure is most useful in experimental studies when an accurate standard level is desired Objections to its use in ordinary practice are obvious

At the time of the first examination of the patient the blood pressure of both arms should be taken, since the two may be different In the presence of unexplained high pressure in the brachial artery blood pressure in the legs should be estimated for in this manner conditions such as coarctation of the aorta may be detected

Blood pressure readings vary with age but the often quoted rule that the blood pressure should be 100 plus the age gives values that are too high A systolic pressure consistently above 150 mm of mercury should be considered abnormal at any age The systolic blood pressure readings of normal adults usually vary between 90 and 140 mm of mercury

The diastolic blood pressure is normally between 60 and 90 mm of mercury It can only be determined by the auscultatory method is a much more important reading and shows less variation than the systolic When the diastolic reading of a patient is 120 or over on successive visits the prognosis may be considered poor

The pulse pressure is the difference between the systolic and diastolic pressures It varies between 40 and 70 mm of mercury High values (70 to 120 mm) are found in hypertension and aortic regurgitation Low values occur in aortic stenosis Addison s disease shock and chronic constrictive pericarditis or acute cardiac tamponade from any cause When the pulse pressure varies from beat to beat pulsus alternans may be the cause This is a very important sign that generally points to a poor prognosis However during paroxysmal tachycardias alternation of the pulse does not have the same significance If alternation is observed when the pulse is slow further clinical examination usually reveals other signs of advanced myocardial disease

Blood pressure in females will average 10 mm lower than in males of the same age and build Since sleep causes a fall in the blood pressure the morning readings may be lower than those taken in the evening Respiration and change in position both influence blood pressure to a slight degree

The blood pressure as usually recorded refers to the brachial artery pressure Readings taken at other parts of the body will be found to vary with the size of the vessel and its position Normally the pressure in the femoral artery is greater than the pressure in the brachial The reverse is true in the presence of coarctation of the aorta Changes in outside temperature may affect the blood pressure and the reading may likewise show considerable variation when cold is locally applied This fact has been made use of in the early detection of individuals predisposed to hypertension who usually show an abnormal response in the blood pressure when an extremity is immersed in ice water for a minute (page 298)

The blood pressure may be estimated by the oscillometric method The use of this instrument enables the physician to acquire additional informa

tion in regard to the character of the arteries of the extremities The oscillometer a triangular metal box is fitted with a special cuff containing two rubber bags A dial with a movable needle records the oscillations of the blood vessel wall while a small aneroid manometer registers the blood pressure level at which they appear and disappear The number of spaces covered by the excursion of the needle is charted for each blood pressure level This is known as the oscillometric index

## Venous Pressure

Determination of the venous pressure is not a new procedure Two hundred years ago Hales measured it by connecting a vein directly to a vertical glass manometer tube The amount of attention that has been given to the question of venous pressure in the past decade and the various methods both direct and indirect that have been devised for its determination indicate a renewal of clinical interest in the subject Venous pressure may be measured indirectly by determining the amount of air pressure necessary to collapse a superficial vein or directly by inserting into the vein a needle connected with a manometer The direct method is simpler more accurate and less time-consuming [97] A convenient apparatus (Fig 37A) consists of (1) an 18 gauge needle (2) a special adapter (3) a 20 cc syringe and (4) an upright manometer tube with a 4 mm bore graduated in centimeters It can be readily assembled and the technic of its successful operation is not difficult to learn

Before the venous pressure determination is made the patient should rest in recumbency on an examining table without a pillow for 15 minutes During this time the apparatus may be sterilized in a shallow receptacle in 70 per cent alcohol The patient s arm is prepared in the usual manner with iodine and alcohol and then supported until parallel to an imaginary line in the mid axilla which is approximately the right auricular level A cuff of a blood pressure apparatus is next placed around the upper arm and inflated to 20 mm of mercury to make the arm veins prominent The apparatus is assembled with the needle on one end of the adapter and the syringe on the other The stopcock of the adapter must be kept parallel to the needle (Fig 37B) As the needle enters the vein blood may be drawn into the syringe for the laboratory and the air allowed to escape from the cuff of the blood pressure apparatus The manometer tube is now fitted into the adapter perpendicular to the arm and the stopcock moved to a position where it is parallel to the manometer tube Blood will at once start up the tube When the column has ceased rising the level on the tube is read This is the venous pressure

The venous pressure of normal individuals at rest in recumbency ranges between 4 and 12 mm of water Exercise causes an increased flow of blood to the heart but in the absence of disease the demand is almost immediately met a greater discharge of blood into the arterial system takes place and there is observed only a slight temporary rise in the pressure If advanced heart disease is present, there is a high venous

pressure reading after exercise which if sustained is indicative of impending cardiac failure Departure from the normal venous pressure readings on slight exertion is seen only in cases where the cardiac involvement is obvious Venous pressure readings will not prove valuable in gauging myocardial capacity in patients in whom congestive failure is absent In other words these readings cannot be used as tests for physical fitness

There is no relationship between venous pressure and arterial pressure

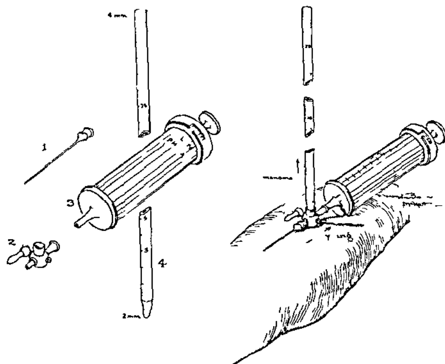



FIG 37 Venous pressure apparatus* A The assembly B Technic of venous pressure determination

in the absence of congestive heart failure The venous pressure readings of 105 compensated cases of hypertension at the Philadelphia General Hospital were found to be within the normal range [97]

Venous pressure estimations are valuable in diagnosis prognosis and treatment of heart disease The first sign of beginning congestive failure is elevation of the venous pressure or venous hypertension In the differential diagnosis of allergic and the so-called "cardiac" asthma the readings are helpful In pneumonia cases in which the physician is ever on the alert for signs of circulatory failure venous pressure readings are valuable since they can detect an early overloading of the right heart

* Made by the George P Pilling and Son Company Philadelphia

Single venous pressure determinations, like single blood pressure readings, are of little value The venous pressure trend, however, is significant If the curve of the readings shows an increasing venous hypertension, this is evidence of the heart s inability to move the blood from the right to the left side and it precedes edema, cardiac dilatation and reduction of urinary secretion If the venous pressure values show a clear-cut upward trend, little hesitancy should be shown in resorting to venesection in order that the right heart may be at least temporarily relieved of part of its burden Readings consistently over 21 mm of water always indicate the need of venesection After this procedure the benefit may be reflected in the lowered values and if sufficient myocardial reserve is present the heart may respond to the decreased load and the circulation will be carried on with a greater degree of efficiency If in spite of venesection, the signs of congestive failure advance and the curve of the venous pressure readings is upward the prognosis is usually poor On the other hand, if the readings show a steadily downward trend clinical improvement may be predicted Under such circumstances venous pressure is valuable in estimating the myocardial capacity of the patient

An estimate of the venous pressure may be obtained in some cases by inspection of the external jugular veins The height of engorgement in the neck veins with the patient in the erect position serves as a rough guide to the height of the pressure within Normally the veins of the neck are never full in this position In congestive heart failure, they may stand out like cords (Fig 51) In some patients the veins of the neck are difficult to see in which event the veins of the hand may be used Normally these veins are collapsed at the right auricular level so observations made on the distance above this level where collapse takes place give a rough estimate of the extent of the elevation of the venous pressure

## ADDITIONAL LABORATORY AIDS IN DIAGNOSIS

**Urinalysis** In addition to the routine urinalysis, a 24 hour specimen should be collected each day and the amount measured and recorded if the patient is suffering from congestive failure The intake-output charts should be kept until circulatory balance is again restored since they enable the physician to gauge the efficiency of digitalis and diuretic therapy

**Routine blood studies** are essential White blood-cell counts are valuable in estimating the severity and extent of the lesion as well as the progress in rheumatic infection subacute bacterial endocarditis and coronary occlusion Serologic studies for syphilis are most helpful in establishing the etiology in doubtful cases A positive serologic finding almost always indicates that syphilitic infection is present However a positive test does not localize the lesion and hence does not prove that the particular disease complex under consideration is luetic Consequently the clinician must evaluate the positive serology in relationship to the whole clinical picture The incidence of false positive results is 1 per cent

or less when a careful worker employs a standard technic and when the following diseases are excluded infectious mononucleosis leprosy, malaria relapsing fever yaws and trypanosomiasis On the other hand, a negative report does not always exclude syphilitic infection because the sensitivity of the best technics in use today is capable of detecting syphilis in only about 80 to 90 per cent of the cases In other words 10 to 20 per cent of syphilitics will yield false negative serologic reports In the latter group of patients a provocative Wassermann test may be helpful

Low Grade Continuous Fever Repeated blood cultures should be made whenever a low grade continuous fever is observed particularly in the presence of an old rheumatic lesion A large amount of blood may be required if organisms in the blood stream are few [09] The most common organism isolated from the blood in heart disease is a member of the *Streptococcus viridans* group When a positive blood culture is obtained the finding should be confirmed with one or two additional cultures One patient brought to my attention had 16 positive cultures over a period of several weeks Unless a special study is being made it is preposterous to inflict so many tests on a patient whose prognosis is usually hopeless

Bacterial Endocarditis It is likewise true that many cases of bacterial endocarditis remain undiagnosed because the disease is not suspected A blood culture which often clinches the diagnosis may not be made because the physician hesitates to undertake a procedure which appears to be outside his field In practice however the taking of a blood culture is about as difficult as taking a blood sample for a Wassermann examination or chemical studies The following technic is recommended *

Prepare a tock of 10 per cent solution of sodium citrate place 0 5 cc of the solution in as many flasks as may be need d Plug with cotton; dry and ster lize the containers in a hot air oven at 175 C for one hour and a half This amount of citrate is sufficient to prevent coagulation of amounts of blood up to 20 cc Apply iod ne and then alcohol over the median basilic vein Under aseptic precautions remove 10 to 15 cc of blood place in the flask and rotate the flask gently to insure uniform d stribution of the anticoagulant The sample is then ready to be s nt to the bacteriological laboratory If the cotton plug is not removed from the citrate flask no contamination will occur for at least two months

**Sedimentation Rate** Many recent studies of the erythrocyte sedimentation rate have demonstrated that this property of the red blood cells is often a valuable guide in the treatment of many forms of cardiac disease [309, 341] Since the technic is simple it will repay the practitioner to add to his equipment the materials needed for carrying out this test

In rheumatic carditis the sedimentation rate is increased when activity is present and tends to become normal with improvement [363] It is useful therefore both in diagnosis and in management In coronary occlusion a decrease in the sedimentation rate indicates healing of the infarcted area [340]

* Suggested by Dr S Brandt Rose Chief Division of Bacteriology Philadelphia General Hospital This method is used routinely for the taking of over 1500 blood cultures per year In the laboratory 2 cc of blood is placed in melted agar and a pour plate is made The remainder of the blood s placed in broth

In these cases the test should be considered along with other factors as a guide to the period of bed rest prescribed

In syphilitic heart disease the sedimentation rate is increased and returns to normal following specific treatment In patients who have aortic regurgitation of questionable origin the test may be of value For example at times we are unable to differentiate clinically between an old rheumatic lesion arteriosclerosis and syphilitic aortitis when aortic regurgitation is noted as the single lesion in a patient in middle life The sedimentation rate is normal in the presence of an inactive rheumatic lesion and arteriosclerosis but will be increased in the patient with active syphilitic aortitis Rapid rates may point to a poor prognosis in syphilitic aortitis whereas a decreasing rate during treatment may be viewed as an encouraging sign

Congenital heart disease if accompanied by cyanosis is associated with an abnormally slow settling time of the red blood cells Normal rates are present in cases showing absence of cyanosis

Congestive Cardiac Failure During congestive cardiac failure arising from any cause the sedimentation rate is slowed The slowing parallels the failure for when balance is restored the rate returns to normal Pulmonary infarcts speed the rate but if they occur in the course of congestive failure the tendency of the latter condition to slow the rate may balance the mechanism and the result may be a normal figure

**Basal Metabolic Rate** Cardiac symptoms and signs accompany both hyperthyroidism and hypothyroidism When the presence of either is suspected a determination of the basal metabolic rate is indicated This is accomplished by a determination of the rate of oxygen consumption Ordinary activity increases the amount of oxygen utilized by the tissues, consequently the patient must be at complete rest for an hour before the determination is made The test is usually carried out in the morning before breakfast since food increases the metabolic rate and gives a false reading The room where the test is made should be comfortable If it is too hot or too cold the rate will be altered

A variety of small portable machines for determining the basal metabolic rate are on the market today Most of them can be relied upon to give satisfactory results if care and tact are used in the preparation of the patient and if ordinary skill is employed by the technician in charge of the apparatus

The basal metabolic rate is an index of thyroid activity When the reading is plus it indicates overactivity while a minus figure indicates underactivity The figure may show considerable variation although over 90 per cent of normal individuals have a basal metabolism between minus 10 and plus 10

Depression of the Basal Metabolic Rate Readings from minus 10 to minus 20 are usually associated with deficiency of thyroid secretion Myxedema states are accompanied by figures from minus 20 to minus 40 per cent Here the clinical picture is typical and in addition the blood cholesterol will be found to be increased (page 373)

ELEVATION OF THE BASAL METABOLIC RATE should be interpreted with care Excitement emotion digestion and fever may give a sizable increase in the absence of thyroid dysfunction In young people with thyrotoxicosis rates as high as plus 70 per cent may be obtained These are usually quickly lowered by rest and iodine (page 357) Essential hypertension or any neurologic condition associated with spasticity or tremor is apt to cause increase in the metabolic rate and care should be used in the interpretation of the readings in these patients Likewise pernicious anemia and leukemia may increase the rate and these diseases although relatively rare should always be ruled out Congestive failure produces an elevation in the basal metabolic rate that according to Harrison[149] is secondary to the labored breathing associated with the dyspnea

Estimation of the basal metabolic rate to be of any value in the diagnosis of thyrotoxicosis should be made by a skilled technician who uses dependable equipment and follows the above mentioned rules More than one initial determination should be made in each case When frequent determinations are impossible in following the progress of a patient the basal pulse rate may be used as a guide to the metabolic rate until the test is made When the patient is at complete bed rest and the pulse rate is 80 or below there is seldom present any great increase in the metabolic rate Basal pulse rates over 100 are usually associated with elevation of the basal metabolism Read s formula in these cases may be helpful B M R = 0 683 [pulse rate + 0 9 (pulse pressure)] − 71 5 For example if the pulse is 100 and the pulse pressure 60 the B M R would be 0 683 (100 plus 0 9 (60)) minus 71 5 equals plus 34 per cent

## NOMENCLATURE OF THE AMERICAN HEART ASSOCIATION

In order that advances may be made in the study of any disease it is essential that workers in various parts of the country use the same terms in designating the same conditions After establishing this uniformity in nomenclature suitable criteria should be adopted for each entity To meet this need the Committee on Cardiac Clinics of the Association for the Prevention and Relief of Heart Disease the predecessor of the Heart Committee of the New York Tuberculosis and Health Association in 1923 prepared a nomenclature covering the diseases of the heart and blood vessels which was introduced into various clinics With certain changes this nomenclature was adopted by the American Heart Association and since this time has been used with success in all parts of the country

Later another committee appointed by the Heart Committee drew up and published an extremely practical guide containing all the criteria for the diagnosis of heart disease Revisions in 1932 and 1939 have increased the usefulness of this small volume* Its contents should be familiar to every physician who undertakes the treatment of cardiac patients

* Heart Disease Distributed by the American Heart Association 50 West 50th Street New York N Y

A glance at the most recent edition will show that the diseases of the heart are considered to include not only the structural changes found in the heart, pericardium and the adjacent structures but also disturbances of function As a result, this broader definition of disease brings within the scope of the nomenclature a number of disturbances that formerly had to be dismissed as functional even though their consequences might have been definitely disabling The usefulness of the nomenclature is increased by this change

The completed diagnosis of every cardiac patient should include one or more titles from each of the main headings of this nomenclature (page 66) There is first a statement concerning the etiology of the disease If structural changes are discovered these should be named or it should be stated that there is no structural alteration Under Part C the cardiac mechanism and any disturbances of cardiac physiology present are included A diagnosis of the cardiac functional capacity and a statement of the patient s therapeutic classification complete the list It is obvious that this comprehensive diagnosis depends on a careful consideration of every aspect of each case and affords a sound basis for management

Certain patients may have symptoms or abnormal physical signs and yet it may not be possible to make a diagnosis of structural disease or any disturbance of cardiac physiology These patients should be re examined at some future date At the time of the examination they are listed as Possible Heart Disease Patients who have no structural defects or physiologic disturbances of the heart but who have another disease capable of causing heart disease may be retained for further observation with the diagnosis of Potential Heart Disease and a statement of the possible etiologic factor

ETIOLOGIC DIAGNOSIS*

1 Anemia
2 Arteriosclerosis
3 Bacterial infection (specify if possible) [1]
4 Congenital anomaly
5 Effort syndrome (neurocirculatory asthenia)
6 Hypertension
7 Hyperthyroidism [1]
8 Hypothyroidism
9 Neoplasm
10 Other etiologic factor (to be specified)
11 Psychoneurosis
12 Pulmonary disease (to be specified)
13 Reflex action
14 Rheumatic fever [1]
15 Syphilis [1]
16 Thoracic deformity
17 Toxic agent (specify if possible)
18 Trauma
19 Unknown

[1] When one of these diagnoses is made it should be stated if possible whether the etiologic factor is still active or inactive

* Reprinted by permission of N Y Tuberculosis and Health Ass n

## ANATOMIC DIAGNOSIS

### Diseases of Aorta and Pulmonary Arteries

1 Aneurysm (specify location)
Aortitis
3 Arteriosclerosis of aorta
a Without dilatation
b With dilatation
4 Arteriosclerosis of pulmonary arteries
5 Congenital anomaly (specify if possible)
6 Embolism of pulmonary arteries
7 Injury of (specify location)
8 Other diseases of aorta (specify lesion)
9 Other disease of pulmonary arteries (specify lesion)
10 Rupture (spontaneous)
11 Thrombosis of aorta
1 Thrombosis of pulmonary arteries

### Coronary Arteries

13 Arteriosclerosis of coronary arteries
a With narrowing
b With occlusion
*14 Arteritis of coronary arteries*
15 Congenital anomaly of coronary arteries
16 Embolism of coronary artery
17 Injury of coronary artery (specify character of lesion)
18 Other disease of coronary artery (specify)
19 Periarteritis nodosa of coronary artery
0 Stenosis of coronary ostium
1 Thrombosis of coronary artery

### Diseases of Myocardium

(Including Conduction System and Heart as a Whole)

Aneurysm of heart (specify location)
3 Atrophy of heart
4 Congenital anomaly (specify lesion if possible)
25 Degeneration of myocardium (specify type if possible)
6 Enlargement of heart (chambers involved may be specified)
a Dilatation
b Hypertrophy
7 Fatty infiltration of heart
8 Fibrosis of myocardium
9 Infarction of myocardium
a Recent
*b Healed*
30 Injury of heart (specify character of lesion)
31 Myocarditis active
3 Neoplasm of heart (specify type)
33 No structural disease
34 Other structural disease of heart (specify lesion)
*35* Rupture of myocardium (specify location)
36 Thrombosis within heart (specify chamber affected)
*37 Undiagnosed structural disease (specify location if possible)*

### Diseases of Endocardium and Valves

38 Congenital anomaly of endocardium of valves (specify lesion if possible)
39 Endocarditis acute bacterial (specify organism)
40 Endocarditis indeterminate
41 Endocarditis subacute bacterial (endocarditis lenta) (specify organism)
42 Injury of endocardium of valve (specify lesion)
43 Mural endocarditis
44 Mural thrombosis
45 Other structural disease (specify lesion if possible)
46 Rupture of valve (specify valve)
47 Sclerosis of valve (specify valve lesion)
48 Undiagnosed structural disease (specify location if possible)
49 Valvulitis active (specify deformity)
50 Valvulitis inactive (specify deformity)
51 Valvular deformity
   a Aortic insufficiency
   b Aortic stenosis
   c Mitral insufficiency
   d Mitral stenosis
   e Pulmonary insufficiency
   f Pulmonic stenosis
   g Tricuspid insufficiency
   h Tricuspid stenosis
52 Calcification of pericardium
53 Congenital anomaly of pericardium (specify lesion)
54 Hemopericardium
55 Hydropericardium
56 Injury of pericardium (specify character of lesion)
57 Neoplasm of pericardium
58 Pericarditis acute
   a Fibrinous
   b Serofibrinous
   c Suppurative
59 Pericarditis chronic
   a Adhesive with constriction
   b Constrictive
60 Pneumopericardium

## PHYSIOLOGIC DIAGNOSIS

### Cardiac Mechanism

1 Arrhythmia (undiagnosed)
2 Auricular fibrillation
   a Paroxysmal
   b Persistent
3 Auricular flutter
   a Paroxysmal
   b Persistent
4 Auriculoventricular block
   a Prolonged conduction time
   b Incomplete
   c Complete
5 Auriculoventricular nodal rhythm (Junctional rhythm)
6 Bunde branch block
7 Other arrhythmias (specify)

8 Paroxysmal tachycardia
  a Auricular
  b Auriculoventricular nodal (Junctional)
  c Ventricular
  d Unknown origin
9 Premature contractions
  a Auricular
  b Auriculoventricular (Junctional) nodal
  c Ventricular
  d Unknown origin
10 Sinus arrest
11 Sinus arrhythmia
1 Sinus bradycardia
13 Sinus rhythm normal
14 Sinus tachycardia
15 Ventricular escape
16 Ventricular fibrillation
17 Wandering pacemaker
18 Valvular incompetence
  a Aortic incompetence
  b Mitral incompetence
  c Pulmonic incompetence
  d Tricuspid incompetence
19 Adams Stokes syndrome
20 Anginal syndrome
1 Cardiac insufficiency
Carotid sinus syndrome
3 Pulsus alternans
4 Paroxysmal dyspnea
25 Paroxysmal pulmonary edema

## FUNCTIONAL CLASSIFICATION

At the present time there is no clinical test which will measure accurately the functional capacity of the heart. For the purpose of this classification it is to be estimated by appraising the patient's ability to perform physical activity. The estimate is only approximate for it is derived largely by inference from the history. It represents an expression of opinion concerning the functional capacity of the patient as modified specially by his cardiac disease.

The diminution in functional capacity which results from a cardiac disorder may be accompanied by discomfort or signs of impaired circulation or both. The extent to which physical activity is curtailed and the severity of the symptoms caused by effort are helpful in estimating the degree of reduction in functional capacity. Usually structural changes are present in the heart. Occasionally, as for instance in certain cases of auricular fibrillation or paroxysmal tachycardia or in certain patients suffering from the anginal syndrome, no anatomic lesions can be detected.

Functional capacity is usually limited because of (1) cardiac insufficiency (2) the anginal syndrome. Other less common causes of limitation are paroxysmal tachycardia and complete heart block. Physical signs may

### Diseases of Endocardium and Valves

38 Congenital anomaly of endocardium of valves (specify lesion if possible)
39 Endocarditis acute bacterial (specify organism)
40 Endocarditis indeterminate
41 Endocarditis subacute bacterial (endocarditis lenta) (specify organism)
42 Injury of endocardium of valve (specify lesion)
43 Mural endocarditis
44 Mural thrombosis
45 Other structural disease (specify lesion if possible)
46 Rupture of valve (specify valve)
47 Sclerosis of valve (specify valve lesion)
48 Undiagnosed structural disease (specify location if possible)
49 Valvulitis active (specify deformity)
50 Valvulitis inactive (specify deformity)
51 Valvular deformity
  a Aortic insufficiency
  b Aortic stenosis
  c Mitral insufficiency
  d Mitral stenosis
  e Pulmonary insufficiency
  f Pulmonic stenosis
  g Tricuspid insufficiency
  h Tricuspid stenosis
52 Calcification of pericardium
53 Congenital anomaly of pericardium (specify lesion)
54 Hemopericardium
55 Hydropericardium
56 Injury of pericardium (specify character of lesion)
57 Neoplasm of pericardium
58 Pericarditis acute
  a Fibrinous
  b Serofibrinous
  c Suppurative
59 *Pericarditis chronic*
  a Adhesive with constriction
  b Constrictive
60 Pneumopericardium

## PHYSIOLOGIC DIAGNOSIS

### Cardiac Mechanism

1 Arrhythmia (undiagnosed)
2 Auricular fibrillation
  a Paroxysmal
  b Persistent
3 Auricular flutter
  a Paroxysmal
  b Persistent
4 Auriculoventricular block
  a Prolonged conduction time
  b Incomplete
  c Complete
5 Auriculoventricular nodal rhythm (Junctional rhythm)
6 *Bunde branch block*
7 Other arrhythmias (specify)

**Class III** (formerly II B) PATIENTS WITH CARDIAC DISEASE AND MARKED LIMITATION OF PHYSICAL ACTIVITY They are comfortable at rest Discomfort in the form of undue fatigue palpitation dyspnea or anginal pain is caused by less than ordinary activity

**Class IV** (formerly III) PATIENTS WITH CARDIAC DISEASE WHO ARE UNABLE TO CARRY ON ANY PHYSICAL ACTIVITY WITHOUT DISCOMFORT Symptoms of cardiac insufficiency or of the anginal syndrome are present even at rest If any physical activity is undertaken discomfort is increased

## THERAPEUTIC CLASSIFICATION

The Therapeutic Classification is intended to serve as a guide in the management of patients For each class, it gives a prescription for the amount of physical activity which is advised

The functional capacity of the patient does not always determine the amount of physical activity which is permitted For example a child with active rheumatic carditis may not experience discomfort on playing base ball yet the physician knows that rest in bed is imperative * There is frequently a difference between the amount of physical activity which the patient can undertake in terms of his functional capacity and that which he should attempt in order to prevent further cardiac damage and bring about improvement The recommendation as to physical activity is based upon both the amounts of effort possible without discomfort and the nature and severity of the cardiac disorder

### CLASSIFICATION OF PATIENTS

Class A Patients with cardiac disease whose ordinary physical activity need not be restricted

Class B Patients with cardiac disease whose ordinary activity need not be restricted but who should be advised against unusually severe or competitive effort

Class C Patients with cardiac disease whose ordinary physical activity should be moderately restricted and whose more strenuous habitual efforts should be discontinued

Class D Patients with cardiac disease whose ordinary physical activity should be markedly restricted

Class E Patients with cardiac disease who should be at complete rest confined to bed or chair

### POTENTIAL HEART DISEASE†

Patients in whom no cardiac disease is discovered but whose course should be followed by periodic examinations because of the presence or history of an etiologic factor which might cause heart disease The diagnosis in these cases is Potential Heart Disease The etiologic diagnosis should be recorded

* Such a patient would be classified as I E

† There are patients in whom the symptoms or signs though suggestive of cardiac disease do not justify a definite diagnosis and from whom is obtained a history of an etiologic factor which might cause heart disease The diagnosis in such cases is both Potential Heart Disease and Possible Heart Disease

be present or absent but their presence or absence should not influence the rating

The classification of patients according to their functional capacity is not to be used as a guide to formulating a plan for management A therapeutic program particularly with respect to regulation of physical activity, is based on information derived from many sources and is considered in the section on Therapeutic Classification Functional classification should not be influenced by the character of the structural lesion or by judgment as to prognosis Psychogenic disability is discounted For example cardiac functional capacity is not regarded as seriously limited when it is clear that the patient's incapacity is due to a psychoneurotic state, even though organic heart disease is present

In the estimation of cardiac functional capacity the term "ordinary physical activity" is used to describe the actual performance of which each patient was capable prior to the onset of manifest cardiac disease Such factors as the presence of active infection in the heart other acute infectious diseases convalescence from an illness muscular weakness anemia arthritis and psychogenic disability may interfere with judging accurately the capacity for exercise In cases of cardiac disease dating from early childhood a patient s normal functional capacity cannot be estimated In estimating a patient s response to effort a comparison must be made between his ordinary and his present capacity for physical exertion Usually this estimate is based entirely on the history particularly with reference to the patient s symptoms on effort An accurate account of the reaction produced by varying degrees of exertion such as walking on the level or up a grade ascending stairs or running is an essential part of the history In general the more intense the subjective symptoms the more marked are the physical signs of cardiac insufficiency Discrepancies may exist between the number and intensity of the physical signs and the degree of subjective distress on effort This is apt to be true particularly in patients suffering from the anginal syndrome, in whom objective evidences of disease may be slight or absent

If there is doubt as to the rating by the method described direct observation of a patient on performing exercise may be helpful The occurrence of undue dyspnea and the appearance of cardiac pain are of special significance

## CLASSIFICATION OF PATIENTS

Class I PATIENTS WITH CARDIAC DISEASE AND NO LIMITATION OF PHYSICAL ACTIVITY Ordinary physical activity does not cause discomfort Patients in this class do not have symptoms of cardiac insufficiency nor do they experience anginal pa n

Class II (formerly II A) PATIENTS WITH CARDIAC DISEASE AND SLIGHT LIMITATION OF PHYSICAL ACTIVITY They are comfortable at rest If ordinary physical activity is undertaken discomfort results in the form of undue fat gue palpitation dyspnea or anginal pain

the heart There were no symptoms referable to the cardiovascular system The past history was negative for rheumatic infection The family history revealed nothing of importance The exercise tolerance of the patient was excellent

PHYSICAL EXAMINATION revealed the presence of mod rate cardiac enlargement which was confirmed by ro ntgen examinatic n No other evid nce pointing to cardiac disease could be elicited The bloo l pressur weight an l the nutritional history showed no departure from the normal The blood Wassermann was negative The blood count and urinalysis were negative The electrocardiogram was normal

CLINICAL DIAGNOSIS A Etiologic Unknown B Anatomic Cardiac hypertrophy C Physiologic Normal sinus rhythm D Functional Classification Class I Therapeutic Clas ification Class B

# 2

# HEART FAILURE

The foxglove's leaves with caution given
Another proof of favoring Heav'n
Will happily display
The rapid pulse it can abate
The hectic flush can moderate
And blest by Him whose will is fate
May give a lengthen'd day
Withering's *Botany*

## MECHANISMS

The aim of all treatment in heart disease is to prevent or if this is impossible to delay for a considerable period the onset of heart failure The signs and symptoms that point to myocardial weakness may be sudden in onset with congestive manifestations in various parts of the body or they may develop slowly after the functional capacity of the heart has been impaired for a long time In clinical practice heart failure is commonly referred to as 'right sided' or 'left sided' indicating that in the beginning at least, the process is chiefly confined to one ventricle This may be better understood when we consider the fact that in some conditions such as hypertension aortic stenosis or regurgitation, or coronary disease with infarction, a continued strain is placed upon the left ventricle Cardiac hypertrophy follows to enable the patient to combat these abnormalities and *it may develop in the absence of symptoms When this response has reached* its maximum and the burden on the heart is unrelieved failure becomes imminent Dilatation of the left ventricle occurs and the blood received from the right ventricle does not all reach the systemic circulation Consequently an engorgement of the pulmonary circulation results If the failure of the left ventricle takes place suddenly the acute engorgement of the lungs may precipitate a paroxysm of cardiac asthma or paroxysmal nocturnal dyspnea The situation then assumes the nature of an emergency, cough develops with expectoration of frothy bloody sputum and is followed by orthopnea If the onset of failure is slow the right ventricle may be able to meet the demand placed upon it by the stagnation of the blood in the lungs by increasing its work In time this chamber likewise shows hypertrophy If failure of the left ventricle occurs gradually dyspnea will increase, and it will appear on less exertion Tachycardia will rarely be absent Examination at this time will reveal an accentuation of the pulmonic second sound giving evidence of the dilemma

present in the lesser circulation If the pulmonic second sound has previously been normal this finding takes on added significance at a follow up examination The systolic apical murmur that accompanies the increase in the pulmonic second sound generally signifies a functional regurgitation through the mitral valve It is of interest of course to note this murmur but it is much more important from the standpoint of management to study *the heart sounds and to detect a gallop rhythm at the cardiac apex if it is* present Inspiratory rales at the lung bases should be searched for at each examination Another valuable sign of exhaustion of the left ventricle is pulsus alternans which can be demonstrated by the use of the sphygmomanometer (page 53) By this sign early failure of the left ventricle may be revealed and intensive treatment begun

*The most common cause of failure of the right ventricle is previous* failure of the left side of the heart [3,3] Sooner or later following left ventricular failure that is attended by the symptoms just described the right ventricle dilates and fails in which event the characteristic signs and symptoms of this condition are added to the clinical picture Certain types of heart disease may predispose to failure of the right side of the heart for example *mitral stenosis or extensive pulmonary fibrosis where resistance to the blood* flow is materially increased (Table II) In these instances after a long

TABLE II

FACTORS PRODUCING STRAIN ON THE HEART

| PRIMARY LEFT VENTRICULAR STRAIN | PRIMARY RIGHT VENTRICULAR STRAIN | PRIMARY STRAIN ON BOTH VENTRICLES |
|---|---|---|
| Arterial hypertension | Mitral stenosis | Mitral insufficiency |
| Aortic stenosis | Pulmonic valve stenosis | Multiple valvular disease (chiefly aortic and mitral) |
| Aortic insufficiency | Pulmonic valve insufficiency | Severe anemia |
| Infarction of the left ventricle | Pulmonary endarteritis | Arterial hypertension aortic valve disease or myocardial infarction *plus* factors producing right ventricular strain |
| | Organic tricuspid insufficiency | |
| | Marked pulmonary fibrosis | |
| | Marked pulmonary emphysema | |
| PRIMARY STRAIN ON LEFT VENTRICLE AND SECONDARY STRAIN ON RIGHT VENTRICLE | | |
| Factors listed in the first column above *plus failure of* the left ventricle | | |

(After Thompson and White)
Reprinted by permission C V Mosby Co Amer Heart Jour 12 643 (December) 1936

continued strain the time arrives when the blood cannot be efficiently pumped through the lungs There is a damming back into the right auricle with functional regurgitation at the tricuspid orifice and signs of right ventricular failure quickly appear The liver at first bears the major part of the burden and accommodates all the excess blood that is possible In cases where the failure occurs suddenly there is acute liver engorgement the capsule is stretched and pain is usually present under the right costal margin If the stasis takes place gradually the liver enlargement may occur without local pain or tenderness but the other signs of venous engorgement will be apparent in the cord like neck veins (Fig 51) If failure is unrelieved effusions follow with the production of hydrothorax and ascites (See Fig 2) Renal stasis may cause oliguria and albuminuria

While it is valuable whenever possible to consider right and left ventricular failure separately there are instances when both sides fail together An example of this is seen in the sudden appearance of signs of congestion that sometimes accompany the paroxysms of tachycardia or fibrillation which occur in the badly damaged hearts of older people The patients with advanced signs of congestion who are seen by the physician in emergencies often present a picture of the late combination of both types Owing to the predominance in practice of the factors contributing to left ventricular failure (high blood pressure and coronary artery disease) this form is much more apt to be encountered than right sided failure

*There have been two schools of thought concerning the mechanisms be*hind the phenomena of congestive failure The first or back pressure theory I have just reviewed Stokes[3, 5] and later Mackenzie were proponents of the forward failure theory Mackenzie [9] stated that dropsy was due to diminution in the force that propels the blood through the capillaries all the symptoms of heart failure arising because of this impaired blood supply

The recent extensive investigations of Harrison[149] have thrown considerable light on the whole subject of cardiac failure and in my opinion they furnish the necessary proof that the symptoms of congestive failure are not caused by diminished flow to the tissues First of all edema and dyspnea are not usually present in such conditions as shock and hemorrhage which are associated with decreased cardiac output Harrison shows that patients with congestive heart failure have been observed who have normal figures for cardiac output and these figures do not increase when congestion clears Finally he offers in evidence the fact that the therapeutic measures forming the backbone of successful treatment of cardiac failure (venesection digitalis and diuretics) do not cause increase in the output of the heart

## CLASSIFICATION

A better understanding of the various types of cardiac failure and their underlying mechanisms is most essential before therapy can be discussed

The following classification has been proposed by Harrison (Table III)

TABLE III

CIRCULATORY FAILURE

I FORWARD FAILURE

A—Forward Failure of the Peripheral Vascular Apparatus ("shock" "collapse")
  1—Hematogenic ("secondary shock")
  2—Neurogenic ("primary shock")
  3—Vasogenic
B—Forward Failure of the Heart
  1—Sudden Death (Due to Ventricular Fibrillation)
  2—Cardiac Syncope
    (a) Neurogenic
      Psychogenic— simple syncope
        vasovagal syncope
    (b) Reflex
      Carotid Sinus Syncope
      Vago-vagal Syncope
      Oculo-cardiac Syncope (?)
    (c) Cardiogenic
      Adams-Stokes Syndrome
      Syncopal attacks in patients with lesions of aortic valve
  3—Cardiac Collapse
    (a) Marked tachycardia
      Auricular Flutter
      Auricular Fibrillation
      Auricular Tachycardia
      Ventricular Tachycardia
    (b) Acute severe myocardial injury
      Coronary Thrombosis
      Diphtheritic Myocarditis
    (c) Mechanical hindrance to heart
      Cardiac Tamponade (a sudden accumulation of blood or fluid in the pericardium)
      Massive Pulmonary Embolism
      Ball Valve Thrombus obstructing mitral orifice
      Inversion of mitral leaflet

II BACKWARD FAILURE

A—Failure of the Left Side of the Heart
B—Failure of the Right Side of the Heart
C—Failure of Both Sides of the Heart

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The peripheral circulation may fail under a variety of circumstances acute hemorrhage (hematogenic) reflex or psychic disturbances (neurogenic) or following the action of substances like histamine acetyl choline or adenylic acid directly on the vessels (vasogenic) The symptoms that ensue (termed shock or collapse) consist of weakness and vertigo that may be followed by syncope and unconsciousness in severe cases fall in body temperature and sweating No dyspnea appears The patient has a grayish color a rapid feeble pulse a decreasing blood pressure and is able to lie flat in bed Inspection will likewise show that the veins of the neck are not engorged

These symptoms may be caused in a variety of ways For example less blood may be returned to the heart owing to a decreasing venous pressure consequently the output of the heart will be reduced A diminished blood

pressure may also result from the vasodilating influences of various substances acting directly on the peripheral vessels or on the controlling mechanism in the central nervous system

It is essential to recognize peripheral failure when it is present and thus avoid prescribing unnecessary treatment for the heart This is all the more important since recent investigations have pointed out that digitalis may actually be a harmful drug in the treatment of peripheral circulatory failure secondary to acute hemorrhage The emergency treatment in peripheral circulatory failure is discussed on page 511

In contrast to the type of 'forward failure that has its origin in the peripheral circulation (*Table III*), the heart itself may be responsible Here we first consider sudden death that may result when ventricular fibrillation occurs Where sudden forward failure takes place and is followed by recovery it is termed cardiac syncope The condition known as 'simple syncope or vaso-vagal syncope is distinguished from the syncope of peripheral failure chiefly by the slowness of the pulse since tachycardia is the rule in peripheral collapse Again venous distention often follows cardiac collapse while the veins are empty in peripheral failure Carotid sinus syncope (page 378) is one of the possible reflex mechanisms, while the vagus may be responsible in other cases (vago-vagal syncope) Oculocardiac reflexes have also been described by Harrison

More serious lesions in the heart itself (cardiogenic) may be responsible for syncope For example the Adams Stokes seizures that appear in cases of A V heart block are considered under this heading The mechanism and treatment of this type of failure have been described elsewhere (page 405) Cardiac failure or collapse may follow the sudden onset of an ectopic rhythm (page 398) When the burden imposed upon the myocardium by one of these abnormal mechanisms is well tolerated by an elderly patient we may be optimistic in regard to the prognosis It is certainly not worth while to perform additional functional tests in such instances

Acute coronary occlusion may be immediately followed by cardiac collapse if a large area of cardiac muscle is suddenly deprived of its blood supply (page 265) In our ward records we often speak of these patients as shocked However the picture differs from that usually encountered in peripheral circulatory failure in the amount of dyspnea or orthopnea that follows occlusion Likewise in many cases of occlusion rales may appear in the lungs and if the patient survives the appearance of venous congestion also verifies the presence of cardiac failure

Mechanical hindrance to the diastolic filling of the heart may occur acutely when blood or exudate accumulates in the pericardial sac (page 164) or it may follow the development of constricting bands of adhesions in which event the cardiac tamponade may be chronic and progressive (page 181) A large pulmonary embolus may likewise act as an obstruction to the blood entering the lungs and arrest heart action (page 425) Large ball valve thrombi may occlude the mitral orifice and cause sudden cardiac collapse (page 152) A rare accident that may be classified under this head

ing of failure from mechanical hindrance to the cardiac action is eversion of the mitral valve (page 565)

This classification of the varieties of forward heart failure as well as the mechanisms responsible should be familiar to every physician It permits so clear an understanding of the many aspects of the problem that it cannot help but result in better treatment "Backward failure" (the dyskinetic syndrome or failure of the congestive type) will be considered in the remainder of this chapter

The careful examination of the myocardium at autopsy in cases of "backward failure' generally shows no anatomic change The old term "chronic myocarditis so often used in referring to patients with congestive failure following hypertensive heart disease, has been discarded owing to the fact that no fibrosis or inflammatory changes can be found Heart failure is purely a functional condition that is prod ced by exhaustion associated with certain chemical changes in the cardiac muscle fibers These important changes elude the pathologist at post mortem The various conditions that bring about this functional change by the extra burden they impose on the circulation are numerous Included here are long-continued hypertension [illegible] valvular lesions recurrent carditis coronary thrombosis, thyrotoxicosis, pulmonary fibrosis, congenital defects uncontrolled abnormal rhythms [illegible] effusions into the pericardial sac Environmental or occupational factors are likewise important in producing the evidences of failure at an earlier date in some patients than they appear in others Except in [illegible] overwhelming rheumatic infection or in the presence of marked congenital defects backward failure is rarely met in children [illegible] plicates the course of rheumatic heart disease in young adults There is a marked increase in the incidence of congestive failure in pa[illegible] 40 years of age because of the greater number of etiologic agents [illegible] time that are capable of placing the necessary strain on the [illegible]

Sodeman and Burch[349] in a recent study of the precipitat[illegible] congestive failure in 100 consecutive cases found that a d[illegible] be assessed in 52 9 per cent of the instances In the patients [illegible] care who showed a gradual onset of failure without demonstra[illegible] the prognosis was poor Over 95 per cent of this group failed [illegible] sate sufficiently to undertake minimal activity If a cause was [illegible] removed the prognosis they discovered was much improved [illegible] per cent of these instances there was sufficient strength [illegible] at least minimal activity The etiologic diagnosis was [illegible] cant in only one group of patients—those suffering fr[illegible] disease with aortic regurgitation These patients when [illegible] developed were usually unable to regain their bala[illegible] precipitating cause

Any circumstance that places a transient and a[illegible] the circulatory apparatus may act as a precipitat[illegible] children upper respiratory infections leading to [illegible] process are largely to blame Overexertion in any [illegible]

a delicate balance and cause signs of congestion to appear Pregnancy, hemorrhage overeating overindulgence in alcohol or mental shock may likewise *act as precipitating causes In older patients urinary tract in* fections particularly those associated with prostatic obstruction, cause increase in the blood pressure and this may upset the cardiac balance Consequently an attempt should be made in every patient treated for congestive failure to determine the cause of the breakdown Its significance as far as future management is concerned is obvious

**Fever** often accompanies congestive failure In some cases when of slight degree no demonstrable cause may be evident in which event it is customary to attribute the fever to the congestive failure However when the *increase in the temperature is more marked a complication should be sus*pected and confirmatory evidence carefully sought The four most common complications in order of their frequency in a series of cases studied by Kinsey and White[190] were pulmonary infarction pulmonary infection active rheumatic infection and acute coronary thrombosis

## TREATMENT OF CONGESTIVE FAILURE

### General Measures

***Making the Cardiac Patient Comfortable*** *The first indication in the* treatment of congestive failure is to make the patient as comfortable as possible The special bed shown in Fig 38 is ideal but is usually available only for hospital treatment Some supply houses rent these special beds at low figures for patients undergoing treatment at home and at times the problem may be solved in this way If the whole bed is not obtainable, a special spring that fits the bed already in use may be secured

In the absence of special beds the patient can be made more comfortable by discarding the various back rests (usually the canvas type) already in *use and mobilizing the supply of household pillows The large ones are* stacked and one is placed under the knees Smaller softer pillows are convenient for the head and under the elbows If the elbows are protected from the start considerable discomfort will be spared the patient at a later date

When the treatment is carried out at home it will usually be better to use the bedside commode The exertion of getting out of bed to the commode is less than would be expended trying to use the bed pan satisfactorily In an emergency commodes may be constructed easily from an old chair

Large doses of cathartics certainly do not add to the comfort of the patient at the beginning of treatment They are no longer given with the purpose of promoting the loss of large quantities of fluid by bowel for the modern diuretics make this procedure unnecessary Today the patient is fortunate in that he takes his digitalis straight and not mixed with various ingredients of a violent nature The added rest that this affords contributes in no small degree to the chances of recovery Later in the course of treat

ment mild laxatives may be given but it is always well to avoid the effer vescent variety Epsom salts in small doses is the cheapest and best of the group and may be given occasionally in the early morning an hour before breakfast

The fact that the patient has been placed in bed with his position prop erly adjusted does not guarantee complete physical and mental rest Worry emotional excitement and noises will induce a state of continued tension of all the skeletal muscles in many patients Until this is relieved

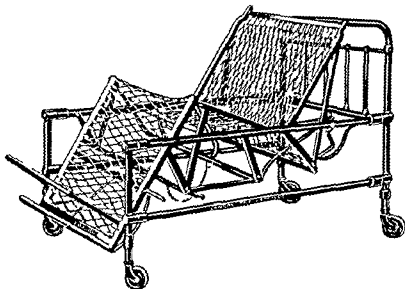


Fig 38 The cardiac bedstead (Redrawn from Diseases of the Heart Sir Thomas Lewis )*

full benefit of any treatment becomes impossible Considerable relief can be gained as Osler stated by the simple process of slowing the body engines While a habit of living cannot be abolished at a command a skillful practitioner soon learns enough about his patient to suggest meas ures that will result in a greater degree of relaxation

The best drug with which to begin treatment of an attack of congestive failure is morphine A hypodermic injection of 15 mg (¼ grain) as soon as the patient is seen and the diagnosis is established acts like a charm It quiets the patient physically and mentally and should never be withheld unless a marked idiosyncrasy to the drug is known to exist Morphine allays dyspnea as well as restlessness and insomnia and during the calm that follows its administration definite plans may be made for the future care of the patient The dose may be safely repeated for the acute episodes of dyspnea until this symptom is controlled since the danger of drug addic

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tion from the use of hypodermics of morphine in the presence of congestive failure is slight if ordinary care is used in each case The fear of addiction should not deprive the patient of the relief that this drug quickly brings to the dyspnea and distress that accompany an attack of congestive failure

When balance is restored restlessness and insomnia may be combated by the use of milder sedatives Phenobarbital may be given in 30 mg (½ grain) doses fortified with 0 7 Gm (10 grains) of sodium bromide after meals If there is restlessness only at night one of the more quickly acting drugs such as sodium amytal may be used Chloral hydrate is now recognized as a safe hypnotic and fear of producing cardiac depression by ordinary doses should not prohibit its use

## DIGITALIS

Although digitalis was known as an ingredient of many family recipes for dropsy before the appearance of William Withering s An Account of Foxglove and Some of Its Medicinal Uses in 1785[418] it remained for this country doctor and botanist to provide the first complete scientific study of the drug (frontispiece) It is unfortunate that in the subsequent years his advice regarding the method of administration of digitalis was entirely *disregarded* and *for well over a century* a *haphazard use of the drug* prevailed Paralleling the revival of interest in digitalis in recent years a vast literature has been built up by the contributions of physiologists pharmacologists and clinicians While many of the beneficial effects of this remedy have been satisfactorily described the final story remains to be written

### Action

The modern concepts concerning the mode of action of digitalis should be thoroughly understood by every physician who prescribes the drug Therefore I offer no apology for including a short review of the present-day belief regarding the mechanism of the principal therapeutic and toxic actions the pharmaceutical preparations dosage and methods of administration indications and contraindications[41] If this shows what appears to be a host of inconsistencies the complex nature of the action of digitalis should be realized However if the different circumstances that are apt to govern the clinical results are kept in mind the reasons for the variations in the action of digitalis will be better understood

Digitalis is a wonderful remedy when correctly administered a useless drug when given in too small amounts and a dangerous poison when the dose is excessive However, a broad zone of therapeutic usefulness exists between the two extremes and the amount of the drug needed to keep the patient in this zone can be gauged by careful clinical observations[6]

The action of digitalis upon the heart consists of two phases The first is a direct effect of the drug on the cardiac muscle including the bundle of His and the Purkinje fibers (modified muscle) The second consists of a stimulation of the vagus nerves for if these are cut the effect is removed

This does not mean that the second effect is necessarily a reflex action Many pharmacologists believe that digitalis stimulates the vagus center It seems certain however that it does not directly stimulate the vagus endings in the heart muscle

**The direct or peripheral effect** of digitalis appears first manifesting itself by an alteration of the T wave of the electrocardiogram This initial indication of the action of the drug may be followed by a predominance of either the vagal or the direct effect

**Vagal Effect** A study of the vagal action which in most if not all respects tends to counteract the direct effects of the drug suggests an attempt on the part of the controlling mechanisms of the heart to compensate for the action of the drug Thus the direct or peripheral action of the drug (1) increases the irritability and muscular tone (2) strengthens the contraction (3) prolongs the refractory phase of the myocardium (4) slows but strengthens conduction by prolonging the refractory phase of the conducting system The vagal stimulation (1) decreases muscular irritability, (2) weakens the auricular contractions (3) shortens the refractory phase and increases the rate but weakens the strength of conduction

In spite of this apparently opposite effect on the part of the vagus both actions coincide in interfering with conduction through the A V bundle and therefore in tending to produce A V block Consequently in auricular fibrillation the ventricular rate is slowed by the direct action of digitalis which prolongs the refractory phase of the bundle of His and by the vagal action which weakens the conduction On auricular fibrillation per se the direct effect of the drug may decrease the rate through the prolongation of the refractory phase while the vagus tends to increase the rate by shortening the refractory period The vagus effect usually predominates in the auricle increasing the rate of the fibrillation Digitalis therefore does not stop the fibrillation but actually makes the auricle fibrillate more rapidly Paradoxically this is beneficial for a speeding of the auricular rate increases the A V block and leads to further ventricular slowing

The late toxic effects of digitalis are characterized by a predominance of the direct action of the drug on the myocardium and the conducting system Consequently atropine is of no value in digitalis poisoning since the main action now is a direct muscular one

If, to the effects mentioned we add the depression of the heart's pacemaker, the sino auricular node that results from the stimulation of the vagus when sinus rhythm prevails we have all the important effects of the digitaloid drugs and the clinical observations can be explained in terms of these actions In therapeutic doses digitalis has no direct effect on the peripheral vascular tree or on the coronary arteries, however, indirectly the coronary flow is altered since the coronaries are of necessity influenced by the rate and the degrees of contraction and relaxation of the heart as a whole

Clinical studies have shown that the administration of digitalis decreases the heart size both in health and in the presence of congestive failure

This effect is consistent and can be demonstrated by careful roentgen ray examinations. Stewart and Cohn[3,4,3] also Harrison and his co workers[10,11] have shown that in health digitalis diminishes cardiac output and minute volume from 20 to 35 per cent while in patients with heart failure improvement is accompanied by a marked increase in the cardiac output. The explanation of this apparent contradiction is to be found in the fact that under normal conditions the heart is at an optimum size for the performance of its work. Digitalis decreases its size according to surface area measurements to three fourths or four fifths of the normal and to that extent lessens its output.

However in the enlarged heart of decompensation, the cavities are dilated but the myocardium is weak, and the output is low. Digitalis through its action on the irritability and contractility decreases the size of the cavities but not to a degree less than normal. Although for the normal heart and for the heart of congestive failure with normal rhythm the increased irritability and contractility and decrease in the heart size satisfactorily explain the results obtained, for the decompensated heart the explanation must never minimize the importance of the drug's effect on decreasing A-V conduction. Indeed the most profound and dependable results from digitalis occur in these cases of auricular fibrillation or flutter with decompensation when the drug decreases A V conduction to a 'toned up' ventricular muscle. Clinical improvement follows the slow rate and increased power of the ventricle regardless of the auricular rate.

The quantitative improvement in the circulatory rate following digitalis may be shown by simple tests based on the length of time required by certain substances injected in the median cephalic vein at the elbow to reach other areas. For example if histamine is used a flush of the face may be watched for (arm to face time) if decholin is used the bitter taste of the drug may be the end point (arm to tongue time), or if ether is injected the appearance of the characteristic odor on the breath may permit a calculation of the arm to lung time to be made. Calculation of the circulation time may be a valuable aid in differentiating cardiac from bronchial asthma when the diagnosis is in doubt. In uncomplicated bronchial asthma the circulation time is normal (page 115).

In man, digitalis produces no important changes in the systolic blood pressure unless the previous level was either high or low and under these conditions the drug tends to bring the systolic pressure back to normal. The increase in the low blood pressure would appear to be the logical sequence of the improved cardiac efficiency. However the fall in the pressure level observed in some cases of the hypertensive type deserves a word of comment. The failing heart in hypertension has produced a deficient circulation, which is accompanied by a poor oxygen supply to the vasomotor center. An attempt on the part of this area to compensate by increased activity causes a rise in the systolic pressure. Digitalis by improving the cardiac efficiency relieves the partial anoxemia of the vasomotor center, which in turn decreases its activity, and the systolic pressure

is lowered The action on the diastolic pressure is more uniform as in the majority of cases it is reduced the net result being an increase in the pulse pressure

### Toxicity

Digitalis should always be prescribed with care Unpleasant effects that are manifestations of beginning toxic action need not attend the successful therapeutic administration of the drug As Withering observed

> The sickness thus excited is extremely different from that occasioned by any other medicine it is peculiarly distressing to the patient it ceases it recurs again as violent as before—(Fig 39)

One hundred and fifty years ago Withering classified this action of digitalis as a direct one upon the stomach mucosa an opinion based upon the fact that digitalis had previously had considerable reputation as an emetic The later demonstration that large amounts of digitalis given by hypodermic injection still produced nausea and vomiting did not convince the physicians of that day that the action was not a gastric one for they then believed that the drug was excreted into the stomach This early toxic action however is on the vomiting center which becomes hyper sensitive to reflexes believed by the author to arise from the heart while others claim that they arise from the abdominal viscera chiefly the liver All preparations of digitalis if they are active and capable of giving good therapeutic results produce nausea and vomiting when administered in large doses

Anorexia and nausea usually precede the vomiting of overdigitalization and these symptoms should serve as a warning The nausea comes in periods or waves that usually disappear within two days after the drug has been discontinued

In rare cases diarrhea may appear as a toxic manifestation of digitalis action while cerebral symptoms should be watched for particularly in older subjects It is important to remember that these signs may occur without the appearance first of nausea and vomiting and are due either to a direct effect on the cerebral centers or to a change in the cerebral circulation If mercurial diuretics are being administered at the same time the issue is further complicated Weiss[394] believes that the onset of the psychosis occasionally seen following the administration of digitalis accompanies circulatory improvement In his opinion the cerebral circulation is already abnormal owing to chronic sclerotic changes and the entrance into it of the toxic products mobilized during the long standing edema causes the onset of the psychosis Headaches as well as confusion may follow digitalis administration the latter progressing in some cases to delirium Loss of memory in older subjects following digitalis is not uncommon The exact cause of disturbances of vision that occasionally follow digitalis is unknown Dimness of vision distortion of colors and rarely blindness may follow large doses of the drug

( 184 )

EFFECTS, RULES, and CAUTIONS.

THE Foxglove when given in very large and quickly-repeated doſes, occaſions ſickneſs, vomiting, purging, giddineſs, confuſed viſion, objects appearing green or yellow; increaſed ſecretion of urine, with frequent motions to part with it, and ſometimes inability to retain it; ſlow pulſe, even as ſlow as 35 in a minute, cold ſweats, convulſions, ſyncope, death.*

When given in a leſs violent manner, it produces moſt of theſe effects in a lower degree; and it is curious to obſerve, that the ſickneſs, with a certain doſe of the medicine, does not take place for many hours after its exhibition has been diſcontinued; that the flow of urine will often precede, ſometimes accompany, frequently follow the ſickneſs at the diſtance of ſome days, and not unfrequently be checked by it. The ſickneſs thus excited, is extremely different from that occaſioned by any other medicine; it is peculiarly diſtreſſing to the patient; it ceaſes, it recurs again as violent as before; and thus it will continue to recur for three or four days, at diſtant and more diſtant intervals.

Theſe

* I am doubtful whether it does not ſometimes excite a copious flow of ſaliva.—See caſes at pages 115, 154, and 155.

Fig 39 Page 184 of William Withering's *An Account of the Foxglove* 1785

of PATIENTS. 191

INFERENCES.

TO prevent any improper influence, which the above recitals of the efficacy of the medicine, aided by the novelty of the ſubject, may have upon the minds of the younger part of my readers, in raiſing their expectations to too high a pitch, I beg leave to deduce a few inferences, which I apprehend the facts will fairly ſupport.

I. That the Digitalis will not univerſally act as a diuretic.

II. That it does do ſo more generally than any other medicine.

III. That it will often produce this effect after every other probable method has been fruitleſsly tried.

IV. That if this fails, there is but little chance of any other medicine ſucceeding.

V. That in proper doſes, and under the management now pointed out, it is mild in its operation, and gives leſs diſturbance to the ſyſtem, than ſquill, or almoſt any other active medicine.

VI. That when dropſy is attended by palſy, unſound viſcera, great debility, or other complication of diſeaſe, neither the Digitalis, nor any other diuretic

Fig 40 Page 191 *Ibid*

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retic can do more than obtain a truce to the urgency of the ſymptoms; unleſs by gaining time, it may afford opportunity for other medicines to combat and ſubdue the original diſeaſe.

VII. That the Digitalis may be uſed with advantage in every ſpecies of dropſy, except the encyſted.

VIII. That it may be made ſubſervient to the cure of diſeaſes, unconnected with dropſy.

IX. That it has a power over the motion of the heart, to a degree yet unobſerved in any other medicine, and that this power may be converted to ſalutary ends.

PRACTICAL

Fig 41 Page 19 *Ibid*

Toxic doses of digitalis cause an increase in the irritability of the myocardium resulting in the production of extrasystoles (more often ventricular than auricular) coupling or pulsus bigeminus (see Fig 248) and various other arrhythmias Large doses of digitalis may be followed by mechanisms of a much more serious nature Auricular fibrillation may appear suddenly Ventricular centers may be stimulated so that they give rise to a succession of impulses resulting in a paroxysm of tachycardia This is extremely dangerous since in some instances it may pass into ventricular fibrillation It is very easy to produce this sequence in the laboratory animal by overdosage of digitalis and it seems reasonable to suppose that the same mechanism produces death in man when care has not been used in the administration of the drug

The effect of digitalis on the sino-auricular node produced principally if not entirely through vagal action results in marked grades of sinus arrhythmia (see Fig 194) and may produce sinus arrest (see Fig 180) Large doses may markedly depress the S A node with the production of a dangerous bradycardia Rarely does the depression of the A V conduction proceed to complete heart block although I have seen this occur several times in exceptional cases following the administration of small doses of digitalis (see Fig 246)

Some investigators have reported an eosinophilia following digitalis administration[344] and claim that this is evidence of an allergic effect Possibly there are allergic reactions to digitalis, but too often the term has been used to cover our ignorance of fundamental reactions and to lull us into a complacency typical of the mind in the Dark Ages I have never seen urticarial rashes or any manifestations that would tend to suggest an allergic reaction Occasionally patients exhibit a hypersensitivity to the drug and show toxic effects following small doses (page 107) However these responses are by no means allergic Patients of this type should be managed without the use of digitalis

To the informed astute observer experience with digitalis in the clinic and at the bedside is the final teacher and as this experience increases it should be possible to employ the drug efficiently with only an occasional need for a cardiologist or the electrocardiograph

### Pharmaceutical Preparations

It is an unnecessary waste of time for the busy practitioner to attempt to familiarize himself with all the different digitalis preparations that flood the market Every one that is fit to use at all has essentially the same action Furthermore many of the proprietary and imported brands are too expensive for the patient in moderate circumstances Since digitalis in many cases once begun will be continued for a long time it is wise to start with a dependable inexpensive preparation This is the U S P whole powdered leaf in capsule or made up in pills or tablets The U S P tincture is equally efficient but it is not as convenient has an unpleasant taste does not keep as well and the dosage is not as easy to regulate The claims

of the representatives of different drug houses that their preparations of digitalis do not produce nausea and lack the bad effects of the numerous other preparations of the drug that patients are in danger of having handed to them across the drug counter as far as I am concerned fall on deaf ears Solubility tests, that salesmen come prepared to perform before my eyes with the premise that the test tube *in the hand represents the stomach* leave me unmoved On occasion I have been informed that I fail to understand and appreciate the great amount of trouble and research that their *company has been through to bring this special preparation of digitalis to* me in its purified state In any event my primary interest is still in the pills made from the U S P whole powdered leaf of digitalis which *assures their potency and the only test I put them to is to make certain* that they can be readily crushed between the fingers If so they are satisfactory It is well to keep in mind that the patient can be saved money when the maintenance dosage has been established by prescribing digitalis in quantities sufficient to last several months Always prescribe digitalis *alone and not in a preparation containing other ingredients for variation* of the daily dosage is difficult in the presence of other drugs Finally attention should be called to the fact that the per Gm potency of the U S P XI digitalis exceeds that of the U S P X preparations by 25 to 30 per cent [88] One hundred milligrams of U S P XI digitalis powder contain one U S P unit of digitalis which is identical with the international unit

## Dosage

The dose of digitalis is *governed by the degree of cardiac failure* and the susceptibility of the patient Eggleston advised one cat unit of digitalis (approximately 80 mg of U S P XI powder or 0 8 cc of U S P XI tincture) for each ten pounds of body weight less the estimated amount of edema fluid present but this amount should be regarded as approximate only It is safe to say that the amount of digitalis whole leaf necessary for the complete *digitalis effect in an adult of average weight lies between* 0 75 to 1 50 Gms (12 to 24 grains) At the first visit I generally prescribe 24 of the 0 1 Gm (1½ grains) tablets of digitalis whole leaf During *the first 24 hours ten of these may be given during the next 24 hours* 0 5 Gm (7½ grains) may be taken The patient then should be carefully watched since the amount that has been given is close to or equivalent to that *needed for the therapeutic digitalization If the occasion is not* an emergency one tablet 0 1 Gm (1½ grains) may be prescribed after each meal for one week and the patient examined carefully at the end of *this time It is not wise to calculate the desired amount of digitalis by* the Eggleston method send the patient away and trust that he will experience no untoward effects until seen again Good treatment today does not consist *in pushing the digitalis until it acts on the kidneys* stomach pulse or bowels and in this respect we are forced to depart slightly from the rules laid down by Withering

*Observation usually tells us when we are reaching the maximal thera*

peutic dose The pulse slows (particularly where fibrillation is present) to 70 beats per minute or less Diuresis appears as the congestive phenomena fade There may be anorexia The amount should then be cut to 0 1 Gm (1½ grains) daily This is the so called maintenance dose and it can in most cases be continued indefinitely Of course, exceptions occur and the exact amount of the maintenance dose will have to be adjusted to the patient by experiment for it will depend on the amount of the drug excreted and destroyed in the body daily For the average patient this figure will be found to vary between 0 06 and 0 18 Gm (1 to 3 grains) Variations occur in each case and the amount may even be different in the same patient under varied circumstances Adjusting this maintenance dose of digitalis is one of the physician s important duties at each follow up examination

Where auricular fibrillation has been present and the rate reduced to 70 by digitalis the patient will be much relieved At this stage of treatment the physician should take the necessary time to explain the aims of therapy and the action of digitalis for it is possible to train the patient to become skilled in the matter of maintenance dosage It is not dangerous to make a student of the pulse out of the patient with chronic auricular fibrillation since much of his future efficiency depends on the successful use of digitalis just as the future of the diabetic depends on the accurate balance between insulin and diet Education of the patient in all matters concerning his disease is invaluable in both instances

The physician should govern the dose of digitalis he prescribes by the interval between visits Large doses should not be given unless it is certain that the physician will go to see the patient the next day Ambulatory patients in clinic or private practice as a general rule should be slowly digitalized

When it is necessary to digitalize a child the amount per pound of body weight in some cases may be found to be greater than that needed for the adult owing to the higher metabolic rate of children This amount has been variously estimated but again EACH CASE SHOULD RECEIVE A DOSAGE DEPENDING ON THE CLINICAL SYMPTOMS AND THE OBSERVED EFFECT OF THE ADMINISTRATION In my experience digitalis does not have the dramatic results in children that are observed in adults This may be due to the rare occurrence in children of auricular fibrillation and flutter

It is just as serious an error to give amounts of digitalis too small to be of value as it is to produce toxic effects While the modern tendency seems to be to prescribe large doses occasional cases are seen in consultation where the doses administered could have no influence whatsoever on the course of the cardiac failure In these instances good results invariably follow increase in the digitalis The administration of too small doses generally occurs in cases where the tincture has been prescribed and the amount to be taken stated in drops Drops will be found to vary according to the type of dropper the temperature the rate of dropping and the

angle of the dropper If the tincture must be used, always have the patient measure the dose in a graduated dropper or medicine glass

INDICATIONS

When digitalis is needed the method of saturation or digitalization that has been described is the only satisfactory way to prescribe the drug When the patient is not showing signs of failure small daily 'tonic' doses have been recommended [7] It may be quite possible to secure tonic effects in patients with organic heart disease of the hypertensive or rheumatic type and in this manner to delay the appearance of dilatation These opinions however are based mainly on animal experimentation and there exists as yet no satisfactory proof that small daily doses in man have the same beneficial action Except in the presence of auricular fibrillation or flutter I have not given digitalis to a patient with organic heart disease in the absence of signs of failure

Rarely is it necessary to employ any other preparation of digitalis than the powdered whole leaf by mouth Sometimes gastro-intestinal disturbances attending congestive failure call for the temporary adoption of another method of administration and in these instances the digitalis may be given by rectum Levy [1] finds digitan an aqueous solution of digitalis to be less irritating to the rectal mucosa than the alcoholic solution of digitalis The equivalent of about 0 1 Gm of the powdered leaf is contained in 1 cc of digitan Consequently following a cleansing enema an amount can be given up to 20 cc through a rectal tube If the digitalis is given in the tincture form it must be well diluted to prevent irritation (approximately one volume of tincture to four volumes of physiologic saline) Absorption by rectum is usually good and the speed of action is comparable with that observed when similar doses are given by mouth

Intramuscular injections of digitalis preparations are painful I quite agree with Fishberg[100] that this route is indicated only when it is impossible to enter a vein

If there is a contraindication to the rectal administration and the emergency is great the intravenous route may be used provided the patient has not been receiving digitalis during the previous week

**Strophanthin** (U S P ) is the preparation of choice for intravenous injection The initial dose should not exceed 0 5 mg (1/120 grain) and 24 hours later 0 3 mg may be given to maintain the effects of the drug When strophanthin is given intravenously effects may be observed within ten minutes It is a good plan when using this method to dissolve the strophanthin in 10 to 20 cc of a 10 per cent dextrose solution and inject slowly As soon as digitalis can be taken by mouth the daily injections of strophanthin are discontinued Brams and his associates[40] have shown however that daily injections of 0 3 mg for as long as 24 consecutive days failed to produce significant clinical or electrocardiographic evidence of toxicity

The pharmacologic properties of strophanthin are identical with those

of digitalis Since strophanthin administered intravenously has a much faster action than digitalis given orally, it has a definite place in emergencies (page 398) In paroxysmal dyspnea and sudden congestive failure excellent effects may be obtained I have seen two dramatic results follow the use of intravenous injections of strophanthin in the receiving ward Both patients were women who developed sudden cardiac failure in the last months of pregnancy and who had not been receiving digitalis previous to admission I have also seen poor results follow the use of strophanthin in some patients who had postoperative tachycardia and in others who had organic heart disease but no evidence of congestive failure

**Ouabain** a crystalline glucoside from g strophanthin is injected in one half the dose recommended for strophanthin The strophanthins are less irritating and less cumulative than digitalis but their absorption is so uncertain that their use by mouth is not advised

**Squill** prepared from the bulb of the sea onion (*Scilla maritima*) has enjoyed a reputation as an efficient therapeutic remedy in a variety of conditions Although employed for centuries in the treatment of dropsy its real value in the management of heart disease and its position as a member of the digitaloid group were factors not fully recognized until 1865 The water insoluble glucosides of squill (scillaren A and B) were isolated in 1934

Recently Chamberlain and Levy [4] have called attention to the use of URGININ which is a mixture of equal proportions of the two active water insoluble glucosides crystalline scillaren A and amorphous scillaren B This preparation can be obtained in tablets containing 0 5 mg (1/120 grain) of the mixed glucosides Assay by the cat method has shown that one of these tablets has an average potency of 2 13 cat units

The results obtained by the use of urginin in cases of congestive failure are similar to those obtained following the administration of digitalis Diuresis is produced and the ventricular rate is slowed particularly in the presence of auricular fibrillation Levy and Chamberlain found that the average total effective dose of urginin is 9 0 mg (18 cat units) and the daily maintenance dose is between 0 5 and 2 0 mg in the presence of auricular fibrillation and 1 5 mg in cases with sinus rhythm Large single doses of urginin are apt to cause gastric disturbances It is best to give 1 5 mg (three tablets) three times daily after meals for two days 1 0 mg (two tablets) twice daily until the desired clinical effects are obtained and then continue with 0 5 mg (one tablet) twice daily as maintenance dose As in the case of digitalis therapy the plan should be changed to fit the individual patient Urginin offers no advantages over digitalis in cases of congestive failure and rectal administration of this preparation is unsuccessful

Urginin has toxic effects similar to digitalis Vander Veer and his associates [378] reported the occurrence of nausea vomiting and diarrhea in patients on maintenance doses of urginin They also observed characteristic toxic arrhythmias ventricular tachycardia coupling of ventricular prema

ture beats and auricular fibrillation These manifestations depend largely on the severity of the cardiac damage and the care with which the drug is given Patients who have slightly damaged hearts may complain of nausea and vomiting before any disturbances in cardiac rhythm are noted On the other hand when severe myocardial damage is present, dangerous arrhythmias may appear before the calculated amount of the drug has been given Although *idiosyncrasy is fortunately rare* we do meet patients at times who exhibit a prejudice against the use of digitalis In these instances urginin may be prescribed

It is well to remember that all digitalis preparations deteriorate with age Powdered leaf of digitalis if kept dry seems to be less affected than other preparations which is another consideration that recommends it for routine use Levy [1] calls attention to the rapid deterioration of aqueous solutions of k strophanthin and of certain digitalis preparations when stored in ampules of soft glass presumably caused by the alkali absorbed from the glass Ampules of hard glass should always be used for these drugs

### CONTRAINDICATIONS

Heart failure is not very often accompanied by situations that contra indicate digitalis therapy There are other conditions however where the use of digitalis in the absence of signs of heart failure may be harmful In cases of partial heart block if there are present seizures that suggest Adams Stokes attacks the drug should not be used It is no longer considered good practice to give digitalis in pneumonia unless the condition of the heart specifically requires it[4, 4] Digitalis may be harmful if given to patients who have angina or coronary occlusion in the absence of congestive manifestations Caution should be used in giving large doses of calcium[38, 133, 3, 3] or ephedrine[1, 4] to patients who are being digitalized Deaths have been reported in cases where intravenous calcium has been given to digitalized patients In diphtheria digitalis should be given with great care since diphtheria toxin and digitalis have somewhat similar effects on the heart

Some surgeons continue to increase the hazards of their anesthesias by giving digitalis routinely by mouth before operation and intramuscularly upon the slightest provocation postoperatively It is without value in the absence of congestive failure pre-operatively and most of the tachycardias I have seen postoperatively arise from shock and other extra cardiac causes where digitalis cannot be expected to be effective and in many instances is actually harmful Except in the presence of a specific cardiac indication digitalis is contraindicated with ether or chloroform These anesthetics increase vagal tone When the augmented vagal activity resulting from digitalis is imposed upon the exaggerated vagal activity of the anesthetic a slight stimulation of the vagi may produce a prolonged diastolic pause If this is preceded by a deep inspiration a fatal exposure of the myocardium to the influence of the anesthetic may be the result

## DIURETIC DRUGS

When the combined measures of bed rest and digitalis do not suffice in removing all edema fluid and restoring cardiac balance, we have at our command a group of drugs whose diuretic properties lend valuable assistance The new members of this group of diuretics have already displayed so high a degree of efficiency combined with so low a toxicity that they are rapidly replacing the older drugs as well as some of the older methods of therapy For this reason Southey s tubes are rarely needed by the modern practitioner and scarification for the relief of extensive edema is seldom required

The diuretic drugs may be conveniently grouped as the xanthines the inorganic salts urea and the organic mercurials

### Xanthines

Members of the xanthine group have a low toxicity and may be administered by mouth but often their action is disappointing The least toxic most efficient and the least expensive member of the group is theobromine sodium acetate It is administered as a tablet or a powder or in a capsule in doses of 0 5 Gm (7½ grains) three times daily

Theophylline with ethylenediamine (aminophylline) is likewise a potent member of this series that can be given orally in doses of 0 1 Gm (1½ grains) three or four times daily or intravenously in doses of 0 24 Gm in 10 cc of physiological saline

Theophylline (theocin) is used in oral doses of 0 2 Gm (3 grains) three or four times daily although it is likely to prove nauseous to some patients

Theobromine calcium salicylate (theocalcin) causes less gastric disturbance and may be given in 1 0 Gm (15 grains) doses after meals

The mechanism of diuretic action of the xanthines may involve several factors the relative importance of which may change under different conditions These factors for which we have at present adequate experimental evidence may be briefly summarized as follows (1) An increased glomerular filtration due to the elevation of the intraglomerular pressure (2) An increase in the number of the functioning glomeruli (3) A decreased tubular absorption (4) An increase in the non-colloidal constituents of the blood

It is best to give a xanthine preparation as long as its effect is maintained in the absence of untoward effects Many clinicians administer members of this group in full doses for three to five days They are then withdrawn and used subsequently as indicated over a similar period

### Inorganic Salts

**Chlorides and Nitrates** Important among the saline diuretics are the chlorides of ammonia potassium and calcium and the nitrates of ammonia and potassium each of which should be given orally The presence of these salts in the blood raises the osmotic pressure of the

plasma with the result that water from the tissues and lymph is transferred more rapidly to the blood transported to the kidneys where it passes with the salts into the glomerular filtrate The tubules refuse to absorb the unneeded salts hence they with the water they withdraw from the tissues pass from the body

When the ammonium salts or calcium chloride are used the typical salt effect is augmented by an acidosis the extent of which will depend upon the amount of the salt administered Theoretical objections can be raised against the use of nitrates which in rare instances may in part be changed to nitrites by the intestinal flora and also to the potassium salts which although entirely nontoxic when administered orally to the normal patient might accumulate in some cardiac cases with decreased renal function in sufficient quantities to embarrass the heart While I consider these effects highly improbable ammonium chloride is the saline diuretic of my choice When administered alone in large quantities ammonium chloride is apt to cause gastric irritability consequently it is better to give it in the form of an enteric coated tablet of 0 5 Gm (7½ grains) Two or three of these administered after meals and at bedtime usually cause no disturbance of digestive function In some patients a daily dose of 10 Gms may be required to produce a satisfactory diuresis

### Organic Mercurial Diuretics

Paracelsus used mercury in the treatment of edema as early as the sixteenth century The success he achieved may be judged when we consider the extensive use of mercury for all diseases in the centuries that followed William Stokes in 1854[3, 8] again called attention to the valuable diuretic properties of mercury when he stated

> It happens again and again that the exhibition of mercury as by enchantment removes the anasarca

Mercury is an ingredient of the famous Guy s pills but its real value as a diuretic was not appreciated until the recent discovery of the less toxic organic preparations In 1920 Saxl and Heilig[2, 8] introduced the first of these compounds known as novasurol (merbaphen U S P ) and verified the earlier statement of Stokes that mercury "as by enchantment removes anasarca About 1927 salyrgan (mersalyl) was introduced on the justification that it was less toxic in laboratory and clinical experiments but the inclusion of merbaphen in the latest edition of the *U S P* is sufficient evidence of the usefulness and safety of the older drugs During the next year (1928) a preparation containing an organic mercurial salt combined with theophyllin was introduced by von Issekutz and von Vegh[1, 1] known as mercupurin All these preparations are ineffective when given by mouth *and must be* administered intravenously or intramuscularly Recently (1954) Engel introduced a suppository containing mercupurin without the theophyllin this is known as *mercurin*[53, 98]

Mode of Action The mercurial diuretics owe their therapeutic value to the fact that they ionize feebly under appropriate conditions to form

mercury ions Although these ions have a general affinity for all proteins their action is manifest principally on the kidney tubules suggesting that here the body presents optimal conditions for the ionization and action of these drugs with the result that reabsorption of the glomerular filtrate is diminished producing a tubular diarrhea

Experiments have demonstrated that the primary site of action of these drugs is not extrarenal If we transplant a kidney of a novasurol treated dog into the neck of a normal dog we will find that the novasurol kidney excretes much more urine than the dog s other kidneys conversely if a normal kidney is transplanted into a novasurol injected dog it will excrete much less than the dog s kidneys which had been exposed to novasurol

TECHNIC OF ADMINISTRATION While three methods of administration have been proposed (intramuscularly intravenously and by rectal suppository) I have obtained the best results from the intravenous route If care is used at all times in making the injections local reactions as well as the sclerosing effects these preparations have on the veins can be prevented LEAKAGE INTO THE SUBCUTANEOUS TISSUES DURING INTRAVENOUS INJECTION SHOULD ALWAYS BE AVOIDED SINCE THESE AREAS ARE SUBSEQUENTLY MOST PAINFUL AND NOT INFREQUENTLY SLOUGH

The equipment necessary for an intravenous injection of any of these mercurial diuretics is simple and may be sterilized and carried to the bed side of the patient in a small container Sterilization of the skin with alcohol is sufficient With the tourniquet in place the needle is held parallel to the veins and inserted When the needle is completely in the vein as shown by the column of blood entering the syringe the tourniquet is released and the injection slowly given In case the veins are deeper owing to the thick subcutaneous tissues a longer needle (1 inch 24 gauge) should be used In patients who have excessive edema or obesity and when other points of entry are obscured the injection may be given successfully into a vein on the back of the hand.

The initial intravenous dose of mercupurin or salyrgan should be ½ cc to 1 cc of the 10 per cent solution Untoward reactions are rare and subsequent doses of 2 cc can be administered safely at intervals of three to six days It is usually unnecessary to give the drugs at intervals of less than three days In cases where intravenous injection is impossible the intramuscular route may be used The dosage is the same Intra muscular injections of mercupurin are best given into the upper outer quadrant of the buttock (see Fig 92) using a 2 to 2½ inch needle of 22 gauge Although this method is more painful a similar diuresis is produced

When there is an objection on the part of the patient to parenteral therapy suppositories may be tried although I have not observed as satis factory results following their use as reported by others[110] However in cases where a good diuretic response follows suppositories have a distinct advantage since it is then possible to give up the intravenous route— always a desirable step when another avenue of administration proves as effective The use of suppositories means fewer visits of the patient to

the clinic or physician's office and consequently results in a saving of time and money. The suppositories are made of a cocoa butter base, and contain 500 mg of the mercurial salt of mercupurin without the addition of theophyllin. The suppository should always be inserted in the morning after a cleansing enema in which event absorption may be fairly rapid. Diuresis starts in one to two hours. The use of suppositories should be guided by the physician depending on the amount of edema present and its tendency to recur. Intervals of four to six days are usually recommended.

The acid producing salts are weaker diuretics in themselves but have been found to augment considerably the diuresis if administered with the organic mercurial diuretics. Keith, Barrier, and Whelan[18] first used ammonium chloride in combination with novasurol and obtained good results when either preparation given alone was ineffective. The mechanism of this synergistic action is probably related to the pH of the glomerular filtrate. Other acid forming salts like ammonium nitrate and calcium chloride were found to be just as effective as ammonium chloride. Administration of alkalis has been shown to reduce the diuresis. Whenever possible the acidifying salt ammonium chloride is given in 1.0 Gm. (15 grains) doses after meals and at bedtime for two days before the administration of the mercurial diuretic.

UNTOWARD EFFECTS. The addition of these powerful agents in the form of mercurial diuretics to our therapeutic program has been a great advance. However, caution should be used in their indiscriminate use, particularly in older people. Too rapid dehydration in some instances has been followed by extreme weakness and not uncommonly by changes in the mental condition of these patients. They lose interest, take little food and may at times show a tendency to an increasing stupor leading eventually to coma. The enormous loss of sodium chloride and water from the body must be considered as one of the dangers of this form of therapy.[30] In addition to the weakness and thirst complained of by the patient, dry tongue, sunken features and other signs of dehydration should be looked for. At times the chloride loss with its accompanying weakness simulates Addison's disease where a chloride deficiency likewise occurs.

The sodium ion is linked with the fundamental biochemical processes of life and one of its chief roles is to hold water in the intercellular tissue spaces. The administration of one of the diuretic drugs causes the loss of sodium chloride from the tissues, the amount depending on the dose of the drug. Consequently when the symptoms described above appear in any patient receiving mercurial diuretics the usual routine therapy should be interrupted, and water and sodium chloride administered by mouth or intravenously as the occasion demands.

In the treatment of congestive failure it is well to remember that all the procedures recommended should not be carried out during too short a period of time, for example thoracentesis, abdominal tap, diuresis and phlebotomy. This is particularly true in aged patients. I have seen weakness and collapse follow "too much doctoring," a state of affairs similar to

that produced by the repeated bleedings of our fathers of old' It is wise to make haste slowly and not try all the drugs in the saddle bag during the first 24 hours

It is not my purpose to arouse any apprehension on the part of the physician in regard to the mercurial diuretics No great concern should attend their daily use in practice when definitely indicated if the above precautionary measures are followed In very sick patients particularly those who suffer from attacks of paroxysmal cardiac dyspnea (cardiac asthma) all intravenous injections should be made slowly It might be well in some cases to dilute the drug to 10 cc with normal saline solution However if not less than three minutes are allowed for the injection most of the organic mercurial diuretics may be used undiluted in the average patient Injections of mercurials may be given at the usual intervals over the course of many years with no untoward effects as accumulating reports in the literature point out (page 113) Rare cases have shown a renal mercurial poisoning[417] but I have never seen this occur Post mortem examinations of the kidneys of patients who have been receiving many injections show no toxic change [5]

Indications for Mercurial Diuretics Cardiac edema is the chief indication for the use of these drugs At times when the administration of digitalis is not tolerated (page 107) the organic mercurials hold a place of prime importance in the schedule of therapy

In certain types of dyspnea particularly the nocturnal variety much can be gained from the use of the mercurial diuretics These attacks of nocturnal dyspnea which frequently complicate left ventricular failure may be prevented by the routine administration of the mercurial diuretics I have seen this beneficial result occur even when edema was not in evidence (page 109)

I believe that the mercurial diuretic group has likewise helped to provide a lengthened day for the cardiac patient who was previously incapacitated at an earlier date by persisting edema If edema remains after the full exhibition of digitalis in patients with regular rhythm the mercurials form the sheet anchor in therapy in any type of heart disease Mercurial diuretics likewise have a place in the edema of nephrosis where the kidneys can excrete urine of a specific gravity of 1 016 or over and where evidence of acute nephritis is absent

Ascites resulting from both cardiac and noncardiac causes calls for the trial of the mercurial group In these cases the result will usually not be as dramatic as is seen when edema fluid is removed from other locations particularly where the ascites is secondary to hepatic disease After an initial abdominal tapping the routine use of a mercurial diuretic may make the repetition of this procedure unnecessary or it may markedly increase the interval between tappings (Case 7)

Contraindications Hematuria seems to be the sole contraindication to the use of mercurial diuretics according to some authorities [1 3] However caution should always be used in older patients especially in the presence

of cachexia fever enteritis or colitis, and hemiplegia Where local rectal conditions (inflamed or thrombosed hemorrhoids, rectal fissures) are *present the use of the suppositories is contraindicated Care should be* taken to prevent spontaneous redigitalization following administration of mercurial diuretics (page 113)

PREPARATIONS MERBAPHEN U S P or novasurol is a compound of mercury with barbital It contains about 34 per cent mercury and is marketed in ampules containing 1 and 2 cc of a 10 per cent solution

SALYRGAN or mersalyl is a fine crystalline powder containing not less than 37 6 per cent of mercury in poorly ionizable form Ampules containing 1 or 2 cc of a 10 per cent solution are obtainable Salyrgan has been recently modified and now is reported to consist of a 'complex compound of mercury and sodium salicyl allyl amino-o acetate in a five per cent theophyllin solution

MERCUPURIN is marketed in ampules each containing a 10 per cent aqueous solution of the preparation One cc contains the equivalent of about 39 mg of mercury in poorly ionizable form The theophyllin in mercupurin is chemically bound to the mercury atom Mercupurin is supplied in 1 1 cc and 2 2 cc ampules

MERCURIN SUPPOSITORIES Each suppository contains 0 5 Gm mercurin equivalent to about 0 2 Gm of mercury or approximately two and one half times the amount of mercury contained in 2 2 cc of the parenterally administered mercurial diuretics It is supplied in boxes containing five and 25 suppositories

ESIDRONE[380, 381] is a stable, neutral crystalline substance which is easily soluble in water It contains *31 2* per cent mercury in nonionizable form and 28 0 per cent theophyllin which is chemically connected with the mercury atom Generally 1 cc of Esidrone administered intravenously or intramuscularly (never subcutaneously) produces gratifying results although 1 5 cc or 2 0 cc may be administered with comparative safety Esidrone may be given one two or even three times per week It is marketed in ampules of 1 cc (1 cc equals 0 14 Gm Esidrone equals 0 043 Gm mercury) Each ampule contains sufficient amount to allow withdrawal and administration of 1 cc

## MECHANICAL THERAPEUTIC MEASURES

**Venesection** A better understanding of the use of digitalis and the discovery of the effectiveness of organic mercurial diuretics have forced one of the oldest of therapeutic procedures almost into the discard Although previously advised in every disorder to which the human flesh is heir blood letting is now practiced only in the presence of venous hypertension that follows right ventricular failure In these acute episodes the relief obtained following the withdrawal of 500 to 600 cc of blood is at times dramatic

The procedure of venesection itself usually offers no difficulty A needle of large caliber is preferable and care should be used in applying the

tourniquet in order that the blood flow in the artery is not shut off An inflated blood pressure cuff makes a convenient tourniquet for ordinary purposes In emergencies a longitudinal incision into the vein serves just as well as venous puncture The amount of blood to withdraw depends on the clinical condition of the patient and is often not as important as the speed with which it is removed Usually observations of the neck veins furnish a rough gauge of the height of the venous pressure but before venesection is repeated a more exact estimation should be made whenever possible (page 54) Bloodless venesection produced by placing tourniquets

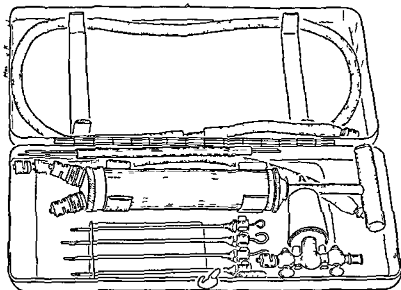
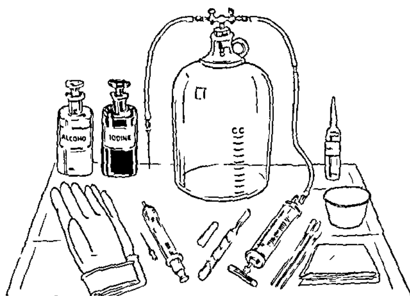


FIG 42 Thoracentesis outfit (Potain)

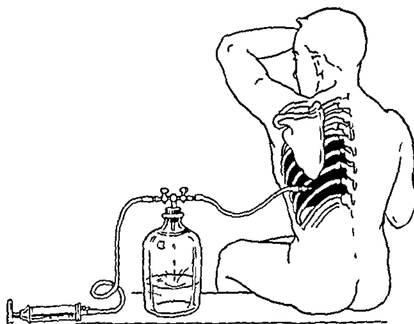
on the arms and legs close to the trunk does not give the same fall in venous pressure in cardiac failure

**Thoracentesis** Digitalis and diuretics although often efficient in preventing the accumulation of fluid in the pleural cavities usually fail to remove large collections present when the patient is first seen These require drainage inasmuch as they reduce the vital capacity and increase the amount of dyspnea Properly carried out thoracentesis should cause the patient very little discomfort

I prefer the Potain apparatus (Fig 42) that enables the removal of the fluid by a closed (siphonage) system A tightly fitting rubber stopcock is essential for success of this method One outlet is attached to the aspirating needle the other to a small suction pump A vacuum is first created in the bottle by closing the stopcock leading to the needle and pumping out the air With the patient sitting up in bed (Fig 43) the site for puncture is




A



B

Fig 43 Thoracentesis
A Essential equipment
B Technic of procedure

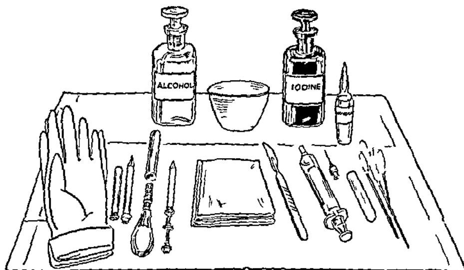
carefully selected (usually the eighth or ninth interspace in the posterior *axillary line) and sterilized* with iodine and alcohol Satisfactory local anesthesia is produced by infiltration of the skin and deeper structures with a 1 per cent procaine hydrochloride solution A small 26 gauge needle should be used for the skin and subcutaneous tissues and then changed to a one and one half inch length of the same gauge when anesthetizing the deeper structures down to the pleura The needle used for the tap should not have too long or too sharp a point in order to avoid puncturing the lung and producing a pneumothorax This danger, with ordinary care is remote A small incision previously made in the anesthetized skin with a scalpel allows an easier and more deliberate entry of the needle into the chest Before inserting the needle the suction in the bottle should be tested by using sterile water taking care to turn the stopcock on the side of the needle to the open position and to close the stopcock leading to the hand pump

The amount that can be safely withdrawn from the pleural cavity varies in each instance As much should be taken as possible without producing untoward symptoms During a thoracentesis the patient should be carefully watched for increase in dyspnea cough faintness or tachycardia Nausea vomiting pulmonary edema and the so-called pleural shock are possibilities but are rare (or else I have been fortunate) When the lung can be felt coming down and striking the end of the needle or if the patient begins *to cough it is well to stop the procedure Withdraw the needle and seal the* puncture with collodion A fall in the venous pressure may indicate improvement even after the withdrawal of as little as 400 cc It is well not to withdraw amounts in excess of 1000 cc at one time

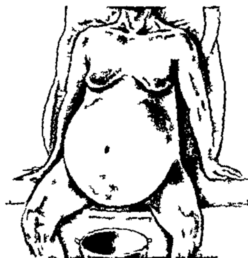
**Abdominal Paracentesis** Small collections in the peritoneal cavity usually show better response to diuretics than thoracic collections owing to the fact that the peritoneum is a better absorbing surface than the pleura An advanced degree of ascites may be present in chronic congestive failure but chronic constrictive pericarditis usually produces the largest collections If the ascites interferes with breathing it should be removed after the pleural collections have been successfully drained

The technic of abdominal paracentesis is simple and there is no contra indication to performing the operation in the home Before proceeding it is essential to have the patient empty the bladder Figure 44A shows the minimum equipment required The operation is carried out best with the patient sitting on a chair or on the edge of the bed A firm satisfactory back rest should be obtained (Fig 44B) The legs are separated and a large rubber sheet or piece of oil cloth fitted around them A household bucket is placed between the feet The skin area below the umbilicus is sterilized in the usual manner with iodine and alcohol (taking care to protect the genitalia) and the site of puncture midway between the umbilicus and the symphysis selected This area is infiltrated with one per cent procaine hydrochloride solution using a 26 gauge needle and followed by injection of the deeper areas using a 24 gauge 1½ inch needle A small incision

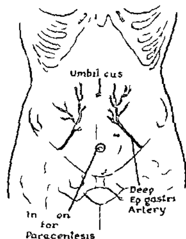
through the superficial tissues is made with a scalpel This point in the technic is important and if carried out will give the patient less discomfort




A



B



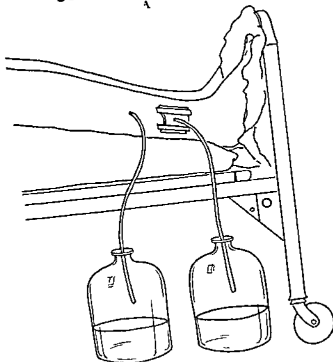

C

Fig 44 Abdominal paracentesis
A Essential equipment
B Position of patient
C Anatomic landmarks

when the trocar is inserted The incision should be large enough to accommodate the trocar which is gradually forced into the abdominal cavity by a series of controlled rotary movements When the cannula is removed



A



B

FIG 45 Southey's Tubes
A Essential equipment
B Tubes in place in subcutaneous tissues

from the inside of the trocar the column of fluid escapes from the end as a jet and can be directed toward the bucket Abdominal paracentesis usually causes no discomfort The withdrawal may be momentarily checked by reinserting the cannula into the trocar taking care to follow aseptic technic continually If the flow of ascitic fluid stops suddenly this same procedure should be employed to displace small pieces of omentum that block the end of the trocar After all the fluid has been withdrawn a sterile dressing is placed over the wound and an abdominal binder applied Following abdominal paracentesis it is a good practice to keep the patient in bed until the next day

**Southey s Tubes** Cases will rarely be met where digitalis and diuretics fail to make an impression on a chronic edema of large proportions In such instances Southey s tubes may be inserted These are small hollow cannulas 5 cm in length and 3 mm in width with a series of openings in the walls (Fig 45) A point on the outer aspect of each leg about two inches above the ankle is anesthetized with procaine hydrochloride and the cannulas are inserted on a small trocar that is withdrawn as soon as the cannulas are in place Sterile rubber tubes leading to bottles attached to the side of the bed are now tied on the end of the cannulas using a fine silk thread The sides of the tubes are kept clean and are surrounded by sterile gauze Occasionally large amounts of fluid (four or five liters in 24 hours) may be drained from the tissues in cases of severe congestive failure by the use of this method

## OXYGEN THERAPY

In the uncomplicated case of chronic cardiac failure oxygen deficiency as determined by an estimation of the degree of saturation of the arterial blood is not present consequently the administration of oxygen is of no value Where there are pulmonary complications (emphysema edema infection infarction) or in cases of coronary occlusion a diminished oxygen saturation may exist in which event the inhalation of high concentrations may be most beneficial It has been shown by Barach and his associates[1 13 16] that oxygen in some cases of cardiac failure relieves the dyspnea and cyanosis slows the pulse promotes diuresis and diminishes edema This effect may be noted three hours after inhaling an atmosphere containing 45 per cent of oxygen Some of these patients when returned to an atmosphere containing a normal amount of oxygen have been reported to show a decreased urinary output and a return of the edema If again placed in the tent a second diuresis often follows As a rule cases of heart failure where an acute rheumatic process exists do not show the same speedy response to oxygen therapy as the arteriosclerotic type

The use of oxygen therapy combined with thyroidectomy for various types of heart disease[17] has been suggested by Barach and his associates Intensive treatment in an oxygen tent preceding and following operation

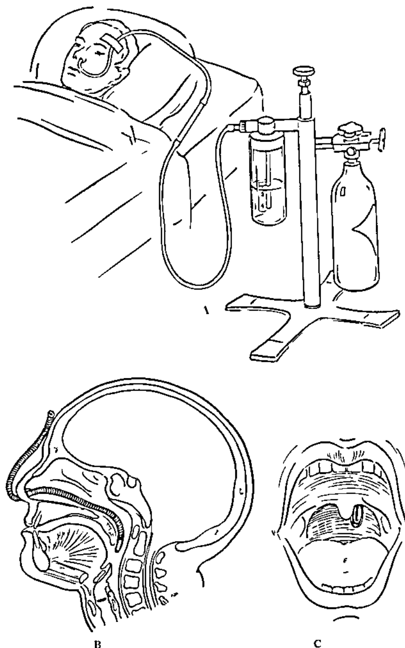



Fig 46 The administration of oxygen by the nasal catheter method
A Bedside apparatus Catheter in place
B Side view showing position of catheter
C Position of catheter in nasopharynx

makes full use of the effect of an atmosphere rich in oxygen on circulatory function

Cyanosis and dyspnea are the most reliable guides to the use of oxygen therapy in the presence of congestive failure with pulmonary complications Cyanosis and cardiac pain serve in the same capacity in cases of coronary occlusion

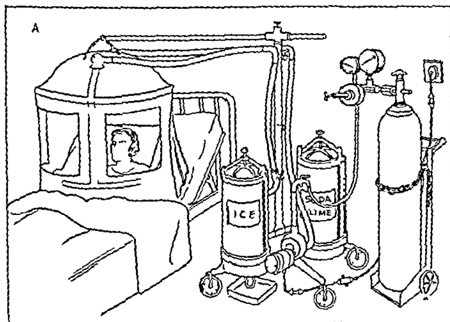
In congestive failure it is my impression that variable results follow the use of oxygen In some cases the cyanosis may clear up but the dyspnea may remain unrelieved while in other cases both symptoms may show a gradual improvement

Levy and Barach[13] have shown that oxygen in acute coronary occlusion is of decided benefit Here inhalation of a 50 per cent mixture supports the heart damaged by anoxemia and consequently relieves the pain A 50 per cent oxygen mixture is recommended in the average case although a 70 per cent mixture can be used if marked anoxemia is present The high concentrations of oxygen (80 to 100 per cent) are apt to cause pulmonary irritation if long continued, while the concentrations below 50 per cent do not have the same beneficial effect

**Methods of Administration** The simplest method to administer oxygen efficiently is by means of the *nasal catheter* The entire equipment consists of a cylinder of oxygen, a suitable regulating valve a humidifier and catheter with connecting tubing (Fig 46A) The supervision requires no special training and the oxygen concentration delivered to the patient compares favorably with the more expensive equipments Care must be taken to place the catheter correctly in the oropharynx Rovenstine[3] suggests that the distance between the external nares and the tragus of the ear (4½ to 5½ inches) be measured on the patient and marked on the catheter before it is inserted A good supply of olive oil or *cottonseed oil is applied to the catheter and with the oxygen flowing* this is introduced slowly through the nares to the mark previously mentioned (Fig 46B) If pushed beyond this point the stimulation produced will usually cause the patient to make swallowing movements The catheter is then withdrawn slightly to the point where deglutition does not occur and fastened in place (Fig 46C) A fresh catheter should be inserted at least every *12* hours and the nostrils used alternately A flow of oxygen of five to six liters per minute gives an alveolar concentration of 50 to 60 per cent With the flow cut down to four liters per minute *a 30 to 40* per cent *alveolar air concentration is obtained Barach believes*[9] *that* the nasal catheter method of administering oxygen in the absence of a tent is the most efficient and recommends a flow of five liters per minute

Various types of small masks for oxygen are on the market [50, 68] The mask designed by Boothby Lovelace, and Bulbulian [6, 43, 2, 8] is efficient and satisfactory and where co-operation can be obtained from the patient most economical (Fig 47C) Enough moisture remains in the mask from the wearer's exhaled air to provide for a sufficiently high and comfortable

humidity The disadvantage of the use of the mask is the interruption necessary for frequent feedings



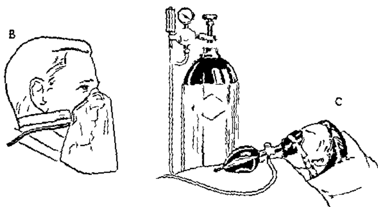




Fig 47 The administration of oxygen
A Oxygen tent
B Small oxygen face tent
C Face mask (Courtesy Ohio Chemical & Manufacturing Company)

A small oxygen face tent has been designed by Barach[10] which is inexpensive and more comfortable than the nasal catheter It is easy to remove and reapply with immediate building up to the oxygen concen-

tration desired The material is transparent and light and may be comfortably molded to the face (Fig 47B)

The administration of oxygen concentrations between 50 and 70 per cent over long periods can be carried out most comfortably and efficiently by the use of the oxygen tent (Fig 47A) The observations of temperature and the humidity that are necessary require the services of a special nurse or attendant consequently this equipment lends itself best to hospital usage In this apparatus the carbon dioxide is removed from the air by soda lime and ice is needed for the cooling system

It has been shown that subcutaneous administration of oxygen does not materially affect the oxygen content or the percentage of oxygen saturation of the arterial blood

Barach[14] has diluted oxygen with helium gas and successfully employed the mixture in patients suffering from severe asthma and in conditions associated with obstruction of the upper air passages (see Chapter 16) Helium is a lighter gas and the helium-oxygen mixture requires considerably less effort to breathe than air and oxygen

A thorough understanding of the technic of the various methods of oxygen administration is essential for success The regulation of the apparatus should never be left to an uninstructed nurse or attendant Physicians unfamiliar with the details of this method of therapy should either not employ it or else should call in consultation a colleague who understands both the errors of technic and the advantages of the procedure In arranging the patient in a tent it is well to keep in mind that the mattress is pervious to oxygen, consequently a rubber sheet should cover the mattress and the patient should be carefully tucked in if the efficiency of the method is to be maintained The tent should be of non diffusing material and should be kept in good condition at all times Leaks reduce efficiency and are expensive It is well to remember that there is only one kind of oxygen used industrial oxygen There is no special 'medical oxygen'

## TOTAL THYROIDECTOMY

Total ablation of the thyroid gland was first proposed by Blumgart and his associates[34] in 1933 as an aid in a carefully selected group of patients suffering from congestive cardiac failure or angina pectoris The scientific basis for this new form of therapy is logical and remains unassailable although the results in many cases have been disappointing

The heart has increased work to do in the presence of an elevation of the basal metabolic rate If this rate is purposely lowered to the myxedema level less work is required of a heart already showing evidence of reaching the limit of its functional capacity by the presence of congestive failure or angina The margin provided by the operation may enable the patient to be about again without recurrence of the symptoms of edema or chest pain on slight exertion Although it was a well known clinical fact that

patients with heart disease and thyrotoxicosis showed considerable cardiac improvement after subtotal thyroidectomy it remained for Blumgart and his colleagues to recommend the removal of the normal thyroid

Patients must be carefully selected not by the surgeon but by the internist only after careful study if successful results are to be obtained Operation should never be considered until a medical regime has been given a fair trial in competent hands Cases are few where digitalis and the use of one of the mercurial diuretics combined with bed rest and diet do not produce marked improvement in all the symptoms of congestive failure The operation is recommended for the small group who remain comfortable under appropriate therapy at bed rest but when the slightest increase in activity is allowed show prompt recurrence of symptoms It must also be kept in mind that the operation that gives this added amount of exercise tolerance is not without its risks where the cardiac lesion has given evidence of its tendency to progress rapidly in patients with coronary arteriosclerosis syphilitic ... complications or in some patients who already have a low basal ... rate nothing is to be gained by attempting thyroid ablation ... it is evident that the number of cases benefited by the ... further decreased White[103] estimates that 1 per cent of all the ... routinely seen for congestive failure or angina pectoris will ... prove suitable for the procedure of total thyroidectomy

In addition to the care necessary in the selection of the ... most important to obtain a surgeon experienced in the ... thyroid operation if fatalities are to be reduced to a ... care of the patient calls for regulation of the myxed... medical regime of digitalis and diuretics is continued as ... is added if required to maintain the basal metabolic ... minus 30 per cent

## FOLLOW UP TREATMENT

When the signs of congestion vanish and the pati... tory the physician s work is by no means finished ... nized very quickly the signs and symptoms of cardia... them promptly and efficiently to the great satisfaction ... his family but the task is not completed until the ... condition has been determined The patient s futu... intelligently planned

In some cases discovery of the etiology will ... others difficult At the time of the initial exam... valuable points may have been gathered fr... example several attacks of rheumatic fever ... tension or angina On examination increase in ... of hyperthyroidism certain characteristic mur... pulsations bulgings or retractions of the thorax ...

ing to the nature of the underlying cardiac lesion may have been discovered (Chapter 1)

When the etiologic background is established the physician is in a position to give advice in regard to the probable future course of the disease For example patients with rheumatic heart disease particularly if auricular fibrillation is present at the onset of congestive failure usually respond promptly to treatment and may show no recurrence of failure for a number of years, provided digitalis and diuretic drugs are properly administered The same may be said concerning cases of hypertension If the initial failure is regarded as a warning and co-operation obtained these patients may do very well for some time The same optimistic outlook cannot be held where syphilitic heart disease is shown to be the cause of the failure Here initial improvement may take place following the usual regime of treatment but it does not last and a poor prognosis must be given The same may be said of patients where frequent coronary occlusions have greatly reduced the myocardial reserve A small number of cases will be seen where prompt recognition of the cause of the congestive failure may result in complete restoration of circulatory function for an indefinite period (pages 182 556)

The type of patient we are dealing with many times has a definite bearing on prognosis If intelligent and co operative the outlook is improved for modern methods of management have much to offer The station in life may likewise affect the future in that the type of work to which the patient returns after the breakdown may precipitate another attack However I do not believe that complete rest for an indefinite period is the best treatment for working people The issue has often been settled for me by the patient when I have been in doubt about the ability to return to a previous occupation Of necessity the head of the house returns to mill or factory, or the housewife to her household duties and the care of her children Mild grades of congestive failure have reappeared in some cases but even so I have been surprised at what could be accomplished by these patients in spite of this handicap

The frequency of congestive failure as a cause of death in heart disease has shown a decided drop during the past decade for the newer methods of treatment enable us to keep cardiac patients edema free Williams and Rainey [403] comparing the incidence of congestive failure in patients from their files who died between 1931 and 1935 with a similar series between the years of 1926 and 1930 found that congestive failure accounted for 31 per cent of the deaths in the early group while it accounted for only 18 per cent in the 1931-1935 series Their figures in addition show that the duration of life after the onset of symptoms has in recent years been distinctly prolonged This reflects the value of the modern treatment of congestive failure

In the following cases problems of management of congestive failure will be reviewed The treatment of the etiologic types of heart disease

*that are represented here has been considered under other chapter headings*

## ILLUSTRATIVE CASES

### *Hypertensive Cardiovascular Disease Complicated by Congestive Cardiac Failure*

**Case 4** Miss E M a *housekeeper of 57*, was first seen 1/ 36 complaining of dyspnea edema and vomiting Four weeks before there was marked increase in dyspnea followed by edema of the feet worse toward evening She was forced to remain in bed *on 1/21/36 because of dyspnea Vomiting began the same day* and when examined the patient was unable to retain even fluids by mouth The past medical history was negative The family history revealed that her mother died at the age of 70 of hypertensive heart disease

Physical Examination BP 130/100 Pulse 130 Rhythm totally irregular Cyanosis and dyspnea were present The heart was enlarged to the left LB 11 6 cm RB 3 0 *cm* There was *a blowing systolic murmur of moderate intensity present* over the mitral area The right chest posteriorly showed flatness as far as the angle of the

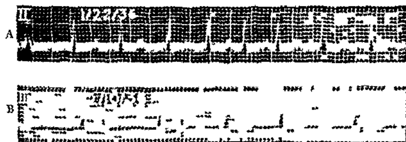


Fig 48 A Auricular fibrillation Ventricular rate 150
B Following digitalization Note the depression of the S T intervals and the greatly reduced ventricular rate

scapula The liver was enlarged 6 cm below the right costal margin No ascites Edema of the legs was present

Laboratory Data The electrocardiogram (Fig 48) showed auricular fibrillation with a rapid ventricular rate Urinalysis light cloud of albumin specific gravity 1 02 no sugar and an occasional red blood cell

Clinical Diagnosis A Etiologic Hypertension B Anatomic Cardiac hypertrophy C Physiologic Auricular fibrillation Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

Discussion This clinical picture is frequently encountered Although the systolic blood pressure was not elevated when the patient was first examined the diastolic level showed a suspicious increase The presence of cardiac enlargement the family history of hypertension and the age of the patient were additional factors of importance in establishing the etiologic diagnosis We may surmise from the history that early left ventricular failure was present four weeks before the patient was compelled to remain in bed Failure of both ventricles was evident when she was first examined

Treatment was begun with an injection of morphine sulfate 15 mg (¼ grain) Because of the cyanosis dyspnea, and tense jugular veins a venesection was performed and 600 cc of blood was quickly withdrawn Following this, the venous engorgement was visibly improved, and the patient felt better

Vomiting was the next problem that demanded attention No medication had been previously taken This removed the possibility of vomiting as a result of digitalis toxic action In patients showing such marked clinical evidence of venous engorgement vomiting usually arises secondary to the congestion of the entire gastro intestinal tract

Since it was impossible to give digitalis by mouth when the patient was first seen the rectal route was chosen After a cleansing enema 8 cc of digitan (equivalent to 0 75 Gm [12 grains] of the whole leaf of *digitalis) were given through a tube and washed in with one ounce of* tap water For the next 12 hours nothing but cracked ice in small quantities was allowed The next day the vomiting had stopped diuresis had started and the patient felt much improved Oral administration of digitalis was therefore begun and one tablet 0 1 Gm (1½ grains) of the whole leaf was given every four hours The pulse on the second day was still irregular, but the rate had dropped to 90 beats per minute A Karell diet (page 548) was prescribed and was well tolerated The vomiting did not recur

Since some edema was still present on the fourth day 1 cc of mercupurin was given intravenously and at the same time the digitalis dosage was cut to 0 1 Gm (1½ grains) twice daily inasmuch as the pulse rate had dropped to 80 The diet was increased and the fluid allowance raised to 2000 cc daily On the sixth day the pulse was 70 consequently the digitalis dose was lowered to maintenance 0 1 Gm (1½ grains) daily The blood pressure at this time was 180/100 confirming the original impression concerning the etiology This increase *was not regarded as an alarming sign The blood pressure* with restoration of circulatory efficiency had merely returned to its previous level

At the beginning of the second week a regular diet was prescribed and the patient was allowed to sit in a chair beside the bed There was no evidence of edema dyspnea did not reappear and the pulse rate was 75 and irregular In the presence of cardiac enlargement and congestive failure, no attempt was made to restore normal rhythm Auricular fibrillation was accepted and the ventricular rate controlled by maintenance doses of digitalis These patients rheumatic or hypertensive get along better with an established fibrillation than they do if attempts are made to restore co-ordinated auricular contractions Restoration of sinus rhythm by quinidine materially increases the possibilities of embolism from clots that may be swept away from the auricular walls

Three weeks later this patient was able to carry out lighter duties of the household The heavier work that had been largely responsible for precipitating the attack of congestive failure was turned over to a part

time maid Rest periods were continued in the afternoon The patient was permitted to take short walks on the level out-of doors on clear days and social activities were encouraged Since she enjoyed sewing and was skilled in certain types of needlework she managed in time to obtain enough work from her neighbors to pay for the services of the maid

### HYPERTENSIVE CARDIOVASCULAR DISEASE CONGESTIVE FAILURE AND CARDIAC ASTHMA IN A PATIENT ABNORMALLY SENSITIVE TO DIGITALIS AND MORPHINE

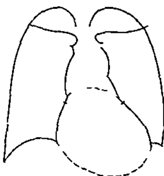
**Case 5** Mrs R M an American housewife of 53 was first seen in January 1937 *complaining of severe chest pain Previously she had received treatment over the course* of five years for hypertension The main symptoms complained of during this time were vertigo and palpitation ( skipping of the heart )

PHYSICAL EXAMINATION When the first examination was made severe chest pain had been present for 4 hours requiring two hypodermic injections of morphine for relief Nevertheless her condition was excellent B P 170/100 T 98 F The pulse was 80 and regular The skin was warm and dry and the color was good An electrocardiogram showed no evidence of coronary occlusion The cause of the chest pain was evident the next morning when a typical herpetic eruption appeared (Fig 49B)

A subsequent study showed that the heart was enlarged to the left (Fig 49A) Systolic murmurs were present over the mitral and aortic areas The aortic second sound was accentuated An electrocardiogram showed frequent premature beats (Fig 49C) and alterations in the T waves consistent with the diagnosis of hypertensive heart disease The blood Wassermann reaction was negative Several blood counts and urinalyses showed no departure from the normal

CLINICAL DIAGNOSIS A Etiologic Hypertension B Anatomic Cardiac hypertrophy Relative mitral insufficiency C Physiologic Frequent premature ventricular contractions D Functional Classification Class 1 Therapeutic Classification Class C

**Discussion** Before the appearance of the skin eruption a diagnosis of acute coronary occlusion had been made This alarmed the patient and the pain that persisted following the healing of the eruption also contributed to a rapid down hill course When re examined three months later the heart was found to be increased in size and dyspnea was present on less exertion Two months later when evening edema began to appear digitalis was prescribed The first dose of the drug consisted of a tablet 0 1 Gm (1½ grains) of the whole leaf This was promptly vomited Repetition of half the dose two days later had the same effect A week later the drug was again administered disguised in a colored capsule This time the vomiting lasted two days and the edema and dyspnea increased to such an extent that bed rest became necessary A capsule containing phenobarbital 30 mg (½ grain) and theobromine sodium *acetate 0 3 Gm (5 grains) given after meals was well tolerated* At the end of a week of complete bed rest the first typical attack of cardiac asthma occurred Although a hypodermic injection of morphine sulfate 15 mg (¼ grain) brought prompt relief it was followed by a severe vomiting that continued for two days When this was controlled attacks of nocturnal dyspnea again returned and were more severe The patient s condition during one of these attacks made the use of morphine again a necessity Vomiting promptly reappeared and the usual measures including gastric lavage were entirely ineffectual in lessening its severity



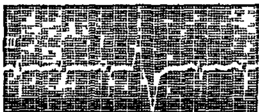
A



B







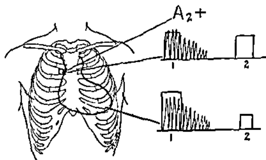
C

FIG 49 A The orthodiagram Note cardiac enlargement (chiefly left ventricular) The right upper cardiac border is formed by the as ending arch Systolic pulsations were visible in this region

B The appearance of the precordial region two days after the onset of chest pain

C The elec rocardiogram *Note* fre quent ventricular premature beats The voltage of the QRS groups is increased T is diphasic and T 3 is flat Left *axis deviation is present*

D Diagram representing clinical findings




D

However the large amount of fluid lost in the vomitus, in addition to the greatly restricted intake during this period appeared to decrease the frequency of the attacks of cardiac asthma Consequently although no edema was visible on physical examination injections of mercupurin were tried Following a satisfactory response to 1 cc intravenously the amount was increased to 2 cc with excellent results Attacks of cardiac asthma did not recur after mercupurin injections were begun

A series of 62 injections of mercupurin have now been given during the past 18 months Although the patient has been confined to her room by the greatly diminished cardiac reserve she has been entirely comfortable and free of paroxysmal dyspnea The only additional medication given during this period has been *ammonium chloride in enteric coated* pills 1 0 Gm (15 grains) after meals and a capsule of phenobarbital 60 mg (1 grain) at night No untoward effects of the mercupurin have been observed The cardiac signs during this time have remained unchanged The rhythm has been more regular and a ventricular rate of 80 has been maintained

This patient is a good example of the progress that has been made in recent years in the treatment of congestive failure In some cases control of the fluid balance of the body through the use of the organic mercurial preparations gives relief even though the accumulation of edema fluid is not evident on clinical examination Future studies are awaited to reveal the mechanism involved in this process

## Syphilitic Heart Disease—Sudden Death During First Attack of Congestive Failure—Autopsy

**Case 6** F R a colored laborer of 40 was admitted to the Philadelphia General Hospital on 5/4/26 The chief complaints were shortness of breath and swelling of the legs and abdomen The patient dated the onset of his illness four months *before admission* when dyspnea on exertion first appeared This increased and was followed by cough Hemoptysis was present on three occasions Six weeks before admission edema of the feet was noted in the evening It gradually became more marked involving the thighs and finally the abdomen The dyspnea had increased to orthopnea on admission The patient gave a history of chancre at the age of 5 for which no treatment had been received

Physical Examination BP 188/ 8 Orthopnea Anasarca Distention of neck veins Pulsating arteries in neck and arms The apex beat was in the fifth interspace in the anterior axillary line Systolic and diastolic thrills were palpable *in the* third and fourth interspaces 1 5 cm to the left of the sternal margin The first sound of the heart heard over the apex was accentuated The aortic second sound was absent and was replaced by a long loud diastolic murmur A loud systolic murmur was heard over the aortic area transmitted to the arteries of the neck Over the third and fourth left inter spaces there was a loud rasping murmur The abdomen was *tense and a* fluid wave was present A pistol shot sound was heard over both femorals

Laboratory Data Wassermann four plus Blood urea nitrogen 75 mg per cent The urine revealed a faint trace of *albumin* The electrocardiogram showed no abnormality except a left axis deviation The roentgen examination showed *cardiac* enlargement with aortic configuration There was no sign of aneurysm

Diagnosis A Etiologic Syphilis B Anatomic Cardiac enlargement Aortic regurgitation Aortitis Aneurysm (?) C Physiologic Normal sinus rhythm Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

A



Fig 50 Syphilitic cardiovascular disease

A Increased cupping of the aortic leaflets is present A sacculation appears behind the anterior leaflet At this site an aneurysm projects into the pulmonary artery

B The Pulmonary Artery The point of rupture of the aneurysmal sac is seen Note the area of thickening on the wall opposite point of rupture (Autopsy No 17 193 Philadelphia General Hospital )

COURSE The patient failed to show any improvement following digitalis and diuretic therapy and died suddenly on the third hospital day

AUTOPSY Marked cardiac enlargement and dilatation were found (Fig 50A) There was increased cupping of all the aortic leaflets The anterior leaflet showed a more marked change with sacculation to the size of a marble in the lower half The base of this sac was ruptured The adjacent tissue of the aortic leaflet was sacculated to the size of a pigeon egg and projected into the pulmonary artery (Fig 50B) The wall of the pulmonary artery opposite the perforation as roughened probably the result of the blood gushing through the opening In the anterior wall of the descending portion of the aorta there was a small sacculated aneurysm containing a moderate amount of laminated clot

**Discussion** The treatment of cardiac failure following syphilitic cardiovascular disease does not differ from that recommended in other types The prognosis however is far more serious in syphilis when symptoms develop to the extent that were observed in this patient on admission Survival of the rheumatic or hypertensive patient for some time following one or more episodes of congestive failure is not unusual On the other hand while a fairly good state of health may be maintained by the patient who has syphilitic aortic regurgitation when signs of circulatory failure can be detected clinically the end of the road is not distant Sudden death may be expected however at any point in the course of the disease Valvular or aneurysmal dilatations with subsequent rupture and the establishment of an intrathoracic arteriovenous aneurysm may precipitate the terminal episode of congestive failure in some instances as demonstrated in this case

Intravenous arsenical preparations are contraindicated in the management of these patients while the heavy metals should be withheld until the evidences of congestive failure disappear This patient received no treatment either for his syphilis or his cardiac condition until three days before death When he entered the hospital the prognosis was hopeless Earlier recognition of the cardiac involvement followed by intensive specific treatment would undoubtedly have delayed the onset of congestive failure many years

### RHEUMATIC HEART DISEASE—STENOSIS AND REGURGITATION AT ALL VALVULAR ORIFICES—CONGESTIVE FAILURE OF EXCEPTIONALLY LONG DURATION

**Case 7** Mrs B K a housewife of 49 when first examined in June 1933 presented the typical picture of congestive failure Circulatory symptoms had appeared four years prior to the initial examination and the patient had been admitted on three occasions to local hospitals for treatment of congestive failure There was a history of three attacks of rheumatic fever in childhood

PHYSICAL EXAMINATION showed a thin adult female sitting up in bed There was a trace of icterus The jugulars were turgid (Fig 51D) Orthopnea was present Inspiratory rales were heard over both lung bases The heart was enlarged to percussion The left border was readily detected in the anterior axillary line and the right border was percussed 6 cm to the right of the midsternal line Systolic thrills were palpable over the aortic pulmonic and mitral valve areas The sounds at the base were replaced by loud systolic and diastolic murmurs Presystolic and diastolic murmurs were heard over the mitral area Separate murmurs were not distinguishable over the tricuspid area Ascites was present (Fig 51C) as well as edema of both feet

LABORATORY DATA The electrocardiogram showed the presence of auricular fibrillation (Fig 51B) The roentgen ray study (Fig 51A) showed enlargement of all chambers of the heart Increased hilum markings were seen but there was no evidence of pleural thickening or effusion Evidence was present of some calcium in the pericardium but fluoroscopically there was good differentiation between auricular and ventricular impulse

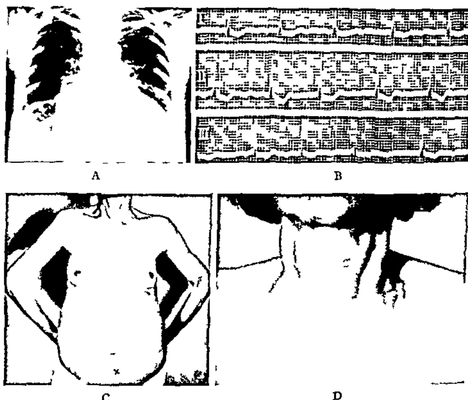


FIG 51 A Roentgenogram showing cardiac enlargement in all diameters
*B The electrocardiogram Auricular fibrillation is present Note the depression of the S T Intervals in all leads caused by digitalis action*
C Marked ascites
D Swollen jugular veins Patient in erect position

indicating the absence of pericardial effusion Urinalysis and blood count were normal The blood Wassermann reaction was negative

CLINICAL DIAGNOSIS A Etiologic Rheumatic Inactive B Anatomic Cardiac enlargement *Mitral aortic tricuspid and pulmonic stenosis and regurgitation* C *Physiologic* Auricular fibrillation Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

DISCUSSION Murmurs and thrills typical of stenosis and regurgitation were elicited over the aortic, mitral and pulmonic areas The findings were not so distinct over the tricuspid area However in the presence of such an extensive rheumatic endocarditis it is reasonable to suppose that the tricuspid valve was involved at least to some degree in the same process The presence of ascites and other signs of venous congestion

gave further support to the diagnosis of tricuspid stenosis Other conditions that might account for the same clinical picture are chronic adhesive pericarditis (page 181), or a massive pericardial effusion Adhesions in the region of the inferior vena cava or at the base of the right lung might obstruct the venous flow with the same result The roentgen study was useful in ruling out these possibilities

When this patient was first seen she was given a hypodermic injection of morphine 15 mg (¼ grain) and digitalized in the usual manner Following the intravenous administration of 1 cc of mercupurin the next day, there was a diuresis amounting to 6000 cc The edema of the feet disappeared completely, and the dyspnea was greatly relieved The ascitic fluid collection was unaffected and two days later 10 000 cc of a clear amber fluid were removed from the abdomen (page 95) Following this procedure the liver was readily palpable a hand s breadth below the right costal margin It was smooth firm and slightly tender

Intravenous injection of 2 cc of mercupurin was begun and continued every five days in an attempt to prevent the reaccumulation of ascites Although the fluid collected in the peritoneal cavity the amount was much less than ordinarily observed and some decrease in the abdominal tension followed each injection of the diuretic During the first few months nausea and occasional vomiting spells appeared the day after the injection of the mercupurin A pulsus bigeminus was observed at this time These signs and symptoms suggested a toxic action following the diuresis which swept back into the blood stream the digitalis contained in the body fluids The maintenance dose of digitalis was accordingly stopped the day before the injections of mercupurin and resumed the following day The toxic effect did not reappear The pulse remained at 70 the appetite improved, and the exercise tolerance was fair A mild state of congestive failure persisted but the patient was able to be about on one floor of the house

During the next six years a total of 430 injections of 2 cc of mercupurin at five day intervals were given to this patient Repetition of the abdominal tapping was unnecessary for four years Paracentesis was performed once during the fifth year and three times during the sixth year The patient is still ambulatory at this writing She has been most co-operative in keeping her intake-output records and is an expert in the matter of digitalis maintenance dosage She illustrates what may at times be accomplished by maintaining digitalis plus maintenance diuretic therapy

Long periods of survival from the time of onset of symptoms of congestive failure are not uncommon in these patients who show combined valvular lesions The continued congestion of the portal system in this case that followed the development of tricuspid stenosis may serve a useful purpose in limiting the return flow of blood to the heart and relieving the strain on a badly damaged organ For the same reason the pulmonary fields show a less marked congestion than is usually found in

patients with mitral stenosis alone Since the left auricle of this patient was greatly dilated it is reasonable to suppose that the mitral stenosis took place first and was followed by the tricuspid lesion at the time of a subsequent attack of rheumatic infection

## Sudden Left Ventricular Failure with Cardiac Asthma in a Patient with a Past History of Bronchial Asthma—Differential *Diagnosis and Management*

Case 8 Mrs L A a housewife of 0 was first seen in May 1939 during an attack of 'asthma The countenance was ashen the lips blue and the skin cold and damp Dyspnea of the asthmatic type was extreme and there were numerous bubbling rales in both lungs and a frothy blood tinged expectoration

Physical Examination B P 1?0/90 The left cardiac border was percussed in the axilla No edema of the extremities was present The liver edge was barely palpable *The past history was negative except for the presence of attacks of asthma for ?0 years* However the usual methods had been unsuccessful in giving relief in the present seizure For this reason the patient was brought to the hospital

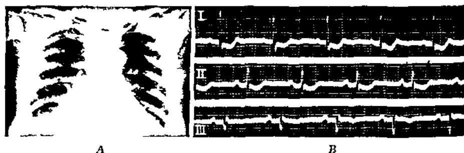



Fig 5 A The roentgenogram shows considerable cardiac enlargement Note calcification of the aorta and the obliteration of the right diaphragmatic angle
B The electrocardiogram shows a deep Q wave in lead 3 and a left axis deviation The S-T Intervals in leads 1 and 2 are depressed (digitalis action)

Clinical Diagnosis A Etiologic Hypertension Arteriosclerosis B Anatomic Cardiac hypertrophy C Physiologic *Normal sinus rhythm Paroxysmal cardiac dyspnea* or cardiac asthma D Functional Classification Class 3 Therapeutic Classification Class E

Discussion The diagnosis of allergic asthma in a patient of this age even in the presence of a history of attacks of asthma for 20 years is risky However if allergic asthma develops late in life (which rarely happens) *it may simulate heart disease* and the attacks may be no different clinically from those observed in patients who have cardiac asthma On the other hand a patient who has had allergic or bronchial asthma for some years may also develop hypertension Hypertrophy of the left ventricle may progress asymptomatically until sudden failure occurs with the production of a more severe type of asthmatic seizure[38] (Chapter 16)

The age the appearance and the size of the heart of the patient under discussion suggested cardiac asthma Likewise the response to rest vene

section and morphine was prompt In other instances however the diagnosis is by no means so easily made

The response to epinephrine in a patient giving a previous history of allergy may be confusing In this case epinephrine had given but slight relief compared to the marked benefit that always followed its administration in previous years Untoward effects on the other hand are not at all uncommon following the persistent use of epinephrine in this type of heart failure

The clinical examination often gives the most valuable evidence for making the differential diagnosis Fortunately in this patient although emphysema was present to some degree accurate determination of the heart size was possible The accentuation of the pulmonic second sound also attracted attention to the congested condition of the pulmonary fields secondary to the sudden failure of the left ventricle In patients who exhibit less pronounced signs of cardiac disease other diagnostic measures are necessary

Venous pressure readings have been useful in differentiating bronchial from cardiac asthma During an asthmatic seizure of purely allergic origin the venous pressure is usually normal while marked elevation may appear in the presence of the left ventricular failure that accompanies cardiac asthma During a paroxysm however the technic of the procedure is not as readily carried out as it is after the attack has been relieved

For this reason estimation of the circulation time is now looked upon as the most valuable laboratory procedure in making the differential diagnosis During an attack of left ventricular failure the velocity of the blood flow through the lungs as well as in the systemic circulation is decreased The cyanide method is probably the best to use because of the sharp end point obtained that does not depend on the response of the patient Arm to lung time may likewise be satisfactorily measured by the injection of 0 3 cc of ether into the arm vein and noting the time that elapses before it is detected on the patient s breath The patient with uncomplicated bronchial asthma will show normal circulation time while cardiac asthma produces an increase in the circulation time [160, 401, 318]

This patient was slowly digitalized after relief of the attack was obtained The whole leaf of digitalis was used and full effect was observed when 15 tablets (0 1 Gm each) had been given over the course of a week At the end of this time the patient felt much improved and since there had been no recurrence of the paroxysmal dyspnea she was anxious to go home Two days following discharge however mild seizures began to appear at night and for the first time a slight amount of edema of the feet was noticed The liver was 5 cm below the right costal margin Consequently she was given two enteric coated tablets of ammonium chloride 0 5 Gm (7½ grains) after meals and injections of mercupurin (2 cc ) every sixth day A maintenance dose of digitalis found to be 1½ grains every second day was continued and 60 mg (1 grain) of

phenobarbital was given at bedtime Although the patient was allowed to be out of bed her activities were restricted to one floor of the house, and she was advised to create a part time position in her household for a willing but much younger neighbor On this program she has had no recurrence of her attacks of cardiac dyspnea for a period of five months

Although a good therapeutic result was obtained considering the age and the amount of cardiac damage present the one mistake in management was made when the patient was permitted to be out of bed and to return home in one week This no doubt, was responsible for recurrence of the attacks A minimum period of three weeks away from even the lighter burdens that arise in the care of a home should be obtained If thrombi have formed in the heart during the period of congestive failure when slowing of the blood stream takes place the prolonged rest period may permit them to become better organized and adherent and consequently there may be less subsequent danger of embolism The longer rest may also be a factor in preventing early recurrence of attacks of congestive failure since it makes possible a more complete training in proper regulation of the regime and allows a better recovery of the cardiac muscle

A patient may occasionally be encountered where the etiology of the congestive failure is obscure If a malignant growth has been discovered in any part of the body, a metastatic process involving the heart should be suspected (page 430)

Edema resulting from vitamin B deficiency states may likewise appear in rare instances in the guise of cardiac failure It may be entirely due to the lack of the vitamin or the vitamin deficiency may be the contributing cause in the appearance of symptoms in a patient with previous cardiac damage Consequently when the etiology of congestive failure is obscure inquiry should be made concerning the dietary habits (page 556)

# 3

# THE PROBLEM OF RHEUMATIC HEART DISEASE

When climatic conditions have augmented the severity of a disease or epidemic the customary therapeutic measures are much less efficacious than a change from the predisposing climatic conditions —CORVISART 1806

Rheumatic infection is the most frequent cause of organic heart disease Appearing in various guises it bewilders us when we become too optimistic concerning our progress in the conquest of infections While perhaps not the most fatal the rheumatic state is one of the most crippling of the diseases that attack mankind Although referred to by some as acute articular rheumatism we can say as we view this infection in the light of modern knowledge that it is neither entirely acute nor entirely articular It now seems certain that once invaded the human host may harbor the inciting agent for months or years and when resistance is lowered acute exacerbations may appear In this respect rheumatic infection is not unlike tuberculosis and syphilis

The rheumatic invasion is not always accompanied by articular manifestations in fact in some instances the disease may be present for years and never produce joint symptoms or for that matter rheumatic pains of any kind As more detailed knowledge is accumulated the real nature of rheumatic disease has been recognized and today instead of considering this modern plague of childhood as an acute disease limited to one region of the body we regard it as a chronic smoldering threat to the integrity of many organs Although much attention centers around the results of rheumatic invasion of the heart few organs of the body fail to feel its touch or lack in a few years the typical scars that reveal its presence The fibrous tissue structures in the body bear the main burden of the attack The heart the joints the subcutaneous tissues the lungs the brain and other organs all show inflammatory reactions that involve this element of their structure

The clinical picture produced by rheumatic infection is by no means uniform In children as a rule the cutaneous tissues are involved to a greater extent than the joint structures However the signs and symptoms may vary even in individuals of the same group One child may have chorea another tonsillitis a third only fever and loss of weight and yet the same amount of heart damage may result in each case

## PRESENT DAY VIEWS ON ETIOLOGY

Although the search has been long and intense the cause of rheumatic fever or the rheumatic state is still a matter of controversy Numerous investigators have isolated streptococci from the lesions, but proof that *these organisms have a direct relationship to rheumatic fever has not been* convincing As early as 1900 Poynton and Paine demonstrated the presence of a diplococcus in the blood and tissues of rheumatic patients Many subsequent investigators have confirmed this observation while others have held out against a too quick acceptance of the streptococcus as the specific cause of the rheumatic process *Shick in 1912* suggested that allergy to the toxin of the streptococcus could account for some of the arthritic and endocardial manifestations of the disease while Schlesinger and his coworkers have considered a filterable virus as the likely cause[3 9] Even protozoa have not been above suspicion as causative agents in rheumatism However while conclusive proof in the matter has yet to be presented a poll of opinions of bacteriologists at the present time would show that the streptococcus leads the list of suspected organisms

**The exact mode of entry** of the causative agent into the body is another unsettled point *Available evidence seems to point to the tonsillar area* as the most likely spot for the invasion to begin since many times initial attacks as well as recurrences are ushered in by the appearance of sore throat Cultures from the tonsillar area during acute attacks often reveal the suspected streptococcus Adenoid tissues and lymph follicles in the pharynx *as well as decayed or abscessed teeth infected sinuses middle* ear disease and the vast area of the gastro intestinal tract have been regarded as possible foci

**Age of Onset** We generally consider rheumatic infection as a disease of childhood or adolescence since in most cases the first symptoms appear between the ages of four and fifteen When the onset is observed in early adult life there are usually differences in the character the manifestations the course and the prognosis In general the earlier the onset the greater the number of recurrences and the greater the chance for cardiac involvement *Females are affected more often than males Wilson and his co* workers[41] in a study of 400 cases of rheumatic infection varying in age from infancy to 22 years found that 61 2 per cent were females and 38 8 per cent males

**Racial Factors** A study of the various races represented in our groups of clinic patients with rheumatic heart disease is misleading since we cannot separate the racial factors from the environmental Beyond a doubt rheumatic infection is widely distributed all over the world and has been observed to attack every race of mankind However, the incidence has been reported to be much less among Chinese

**Disease of Temperate Zone** Rheumatic infection is a disease that

flourishes in temperate climates a fact that should always warrant consideration in the management of these cases Competent observers in tropical climates have repeatedly stated that they have never seen a case of rheumatic fever chorea or mitral stenosis Cold damp temperate climates favor the rheumatic state and in the cardiac clinics situated in large cities along our northern seaboard the bulk of the cases belong to the rheumatic group Acute recurrences are most common among this group in the late winter and early spring months [7 7 79 80]

**Familial Incidence** Rheumatic infection has a tendency to show a much higher incidence in certain families[3] It is not at all uncommon to find a mother and one or two children showing signs of rheumatic heart disease and in some instances the lesions discovered may be identical The practitioner of the old school who acquired his patients at birth and took care of them during the years that followed fully appreciated this tendency for certain types of disease to appear more frequently in some families than coincidence could explain This keen insight often places the general practitioner above the level of the specialist in his ability to detect early manifestations of rheumatic disease and the secret of this skill lies in what we refer to today as hereditary diathesis or to state it his way *some* families offer better soil for rheumatic infection than others

**Environmental influences** cannot be entirely removed from the picture for is it not possible that the rheumatic disease arises from close contacts in families schools or barracks [1 3] On this basis we can also explain its greater frequency in the crowded tenement districts than in the other communities where standards of living are higher Statistics show that rheumatic infection is much more prevalent in cities and towns particularly in crowded industrial sections where it flourishes *among the undernourished* children of the working classes [9 31]

## THE LESION

Rheumatic infection produces a characteristic lesion in many of the tissues of the body We may consider this rheumatic nodule *or Aschoff* body analogous to the miliary nodule that is so common in tuberculosis As it appears in the heart muscle the heart valves the pericardium the brain and the joints it is an example of the proliferative type of tissue reaction The second type of lesion so commonly produced by rheumatic invasion is described as exudative This is found in joints and in the *pericardium* The Aschoff cells that are observed in the proliferative type are large endotheloid cells containing several nuclei They are usually surrounded by lymphocytes and plasma cell and close inspection shows a fibroblastic reaction in the surrounding tissues Aschoff bodies vary in size and shape and are most often situated near a blood vessel (Fig 53)

**The subcutaneous nodule** visible on gross inspection *and so characteristic of* rheumatic disease is similar in its construction to the Aschoff

body (Fig 54) These nodules are generally painless and may be found *in some cases around the malleoli the elbows the knees or vertebral*

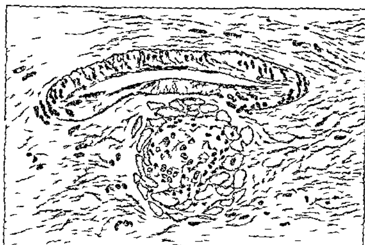


Fig 53 A typical Aschoff body Note relationship to blood vessel

spines *Both of these manifestations of the rheumatic state one occurring* in the myocardium as the Aschoff body of submiliary size and the other visible macroscopically in the subcutaneous tissues may appear and disappear very quickly *Furthermore they may be present in abundance during some years in all rheumatic cases while during other years workers in the same clinic will detect very few This is particularly true of the nodules*



Fig 54 Rheumatic nodules

**Typical Aschoff bodies** are found at autopsy *in many*, but by no means all cases of rheumatic fever They are more apt to be present in the myocardium especially *in the auricular wall and in the interventricular septum* and usually heal with the production of small myocardial scars *Occasionally in severe cases the coronary arteries become involved causing alterations in the blood flow to the heart muscle and consequently grave myocardial impair*ment may result The Aschoff bodies in the septum involve the bundle of His, and the edema that attends the inflammatory process may cause a delay in the transmission of the con

traction impulse from the auricle to the ventricle This will be reflected in the electrocardiogram (page 616) When the acute rheumatic invasion subsides, normal function of the bundle is restored (page 139)

## PATHOLOGY

Rheumatic infection reaches the heart valves through the blood stream Since the mitral valve contains more blood vessels than the others, it is usually the first to be invaded The small vessels in the valves persist longest in the mitral valve Therefore the younger the patient the greater the likelihood of rheumatic involvement In the healing process that follows the delicate texture of the valve is destroyed and it becomes thickened and stiffened Calcium deposits may occur later in these diseased tissues causing them to stand out in the blood stream increasing the obstructive or stenotic nature of the process An equally important feature of rheumatic infection as far as valvular function is concerned is the invasion and subsequent contraction of the chordae tendinae

**The inflammatory changes** produced by the rheumatic process in all severe cases spread readily to the pericardial surfaces The gloss that normally characterizes this epithelial structure quickly disappears and is replaced by a fibrinous exudate The pericardial layers become adherent and when separated at autopsy the typical 'bread and butter appearance is seen Fluid usually accompanies these exudative reactions and accumulates in the pericardial sac in small amounts

**Relation of Structural Changes to Physical Signs** Invasion of the myocardium endocardium and pericardium produces the structural alterations just reviewed These form the basis for the physical signs that we search for in patients suspected of harboring rheumatic infection Various murmurs that have been described previously (page 17) are produced the most common of course, is the one that accompanies mitral regurgitation The stiffening and thickening of the mitral valve causes the presystolic murmur of stenosis at the time of auricular systole which is best heard over the region of the apex beat while weakening of the muscular ring figures largely in the production of the regurgitant murmur When the aortic cusps are invaded insufficiency is produced, and a diastolic murmur appears which is best heard along the left sternal border In rheumatic disease at this area valvular damage is the chief factor in the regurgitation In syphilitic disease regurgitation results when the aortic wall becomes weakened and dilated and when the valve insertions in this area are included in the mass of inflammatory tissue

Invasion of the pericardium with the deposition of fibrin produces a friction rub (page 165) Later adhesions form If the layers of the pericardium become adherent little harm results and no characteristic signs appear However if the thickening of the pericardium takes place and interferes with diastolic filling of the heart much disability may result during the ensuing years (page 179)

CONSIDERABLE MYOCARDIAL DAMAGE may quickly follow acute rheumatic infection and dilatation and failure may take place early Inflammatory lesions in the myocardium near the conduction bundle or its branches may make their presence known by functional alterations Focal myocardial lesions may likewise produce changes in the heart rhythm by initiating premature auricular or ventricular beats

**Other organs of the body** may be extensively involved in the spread of the rheumatic infection Changes in the brain produce the symptoms of chorea and when the lungs are invaded the characteristic hemorrhagic manifestations that often appear have been termed 'rheumatic pneumonia Vascular lesions in the aorta as well as in the small arteries of the body are often encountered Although the damage in the aorta may resemble that produced by syphilis in type, it cannot approach it in severity consequently mycotic aneurysms following acute rheumatic infection, while possible, are rare occurrences

In each patient the extent and severity of the rheumatic involvement will be different Even when the damage is confined to the heart one patient may have a greater degree of myocardial damage with endocarditis and present many signs that can be readily detected clinically while another may have an invasion of the myocardium that will produce few clinical signs and those demonstrable with great difficulty if at all in the early stages

## SIGNS AND SYMPTOMS

The most valuable signs and symptoms to elicit in cases of rheumatic infection are those produced by activity of the process for these guide us in the management of the patient This is particularly true in childhood when the symptoms of the disease often simulate a tuberculous infection

**Loss of Weight** Activity of the rheumatic process is usually attended by loss of weight although it is not as extreme as in tuberculosis Many cases simply do not gain and return to the physician month after month registering the initial weight Very few are overweight Coombs claims that malnutrition is not so definite when aortic lesions predominate[71 7] He attributes the failure to gain weight to the interference with oxygenation of the tissues that is present in mitral stenosis and points to the fact that when this lesion develops early in life grave interference with development may take place (mitral dwarfism)

**Pallor** is usually present, and its appearance should always suggest activity of the rheumatic process It may be accompanied by a moderate secondary anemia and when mitral obstruction is present by a trace of cyanosis as well Marked pallor of a characteristic type is seen in these patients when subacute bacterial endocarditis develops

**Fever** usually attends the course of the disease but its absence should not be considered as a strong point against the diagnosis of an active rheumatic lesion in the presence of other constitutional symptoms The

elevation of the temperature may be very slight and in some cases this may be the only evidence of activity on physical examination High temperatures usually accompany fulminating infections particularly when the brain is the seat of widespread involvement

**Anorexia** In the presence of even slight fever anorexia will appear, the child will show a disinclination to play and will tire easily Irritability and nervousness are common symptoms The first visit to the physician is often made at this stage A careful examination of the run down child may then permit an early diagnosis of rheumatic infection (page 169) Many of these patients are sent to the hospital for tonsillectomy with no other symptoms than pallor anorexia and failure to gain weight A careful study of every child should be carried out before operation since an ill advised tonsillectomy at this stage usually gives a considerable impetus to the invasion

**A sore throat** heralds the onset of a rheumatic exacerbation in many instances, but is not invariably present All types of muscle and joint pains in children should be viewed with suspicion and a thorough investigation should be made The importance of joint pains is emphasized when they occur with any other of the manifestations of rheumatic disease The joint pain may be a mild evanescent process in some cases while in others one joint after another may be attacked in typical textbook fashion usually the wrists ankles knees elbows and shoulders in the order named the pain and swelling disappearing from one joint as another is attacked

**The nervous child** should be examined to rule out rheumatic infection Children sent home from school because of their inability to sit still should be disciplined only after a visit has been made to the physician The twitchings and choreiform movements become pronounced and generalized when the infection in the nervous system has advanced Choreiform movements are accentuated by excitement and activity and are usually absent during sleep Although rheumatic infection is by far the most common type of involvement causing chorea other diseases for example syphilis and encephalitis may occasionally produce it

**Symptoms of a pulmonary nature** may occur in many cases, these have suggested to some investigators a special type of rheumatic pneumonia Specific lesions in the lungs have been described[137 39 56] but here there is much difference of opinion Hemorrhagic manifestations should not puzzle the physician when they occur during the course of acute rheumatic fever When they select the lungs and many alveoli are involved they produce physical signs that simulate pneumonia [393]

**Abdominal Pain** During the course of rheumatic fever particularly in children attacks of abdominal pain are not infrequent In some cases this symptom may be referred from a pericardial lesion In others the pain may be related to the gastric disturbance that sometimes attends salicylate medication In some children suffering from acute rheumatic infection unexplained vomiting may occur Rarely lesions of a rheumatic nature involving the hip joint may cause abdominal pain

**Invasion of the heart** during the course of rheumatic fever may be heralded by an increase in the temperature and an elevation of the pulse rate When the pericardial sac is involved precordial pain may appear It is sharp, increased by breathing and by pressure of the stethoscope over the heart area and is usually accompanied by the characteristic to and fro friction sound In some instances the friction rub is present without pain A dull aching variety of precordial pain may occur at times in the absence of pericarditis Pain of the anginal type is very rare following rheumatic infection except in the presence of aortic valvular disease with regurgitation when it is usually believed to be a result of the low diastolic pressure and the decreased coronary filling I have seen only two rheumatic children who had anginal pain associated with an aortic regurgitant lesion

PALPITATION occurs and may be part of an effort syndrome that not infrequently accompanies any infectious process or it may be the result of the presence of frequent premature beats Only rarely is it caused by paroxysms of tachycardia or auricular fibrillation

DYSPNEA frequently accompanies an acute rheumatic carditis in children and is generally attended by cardiac dilatation It may be accentuated by the collection of fluid in the pericardial sac Very seldom is the fluid present in sufficient amount following rheumatic infection to cause symptoms of tamponade and to require tapping (page 164) Dyspnea is one of the most common symptoms of chronic rheumatic heart disease usually increasing as the lesion develops

**Hemorrhagic episodes** are characteristic features of rheumatic disease consequently an unexplained nose bleed in a child particularly if recurrent is a suggestive symptom Purpura may be a rheumatic manifestation in rare cases although even when associated with joint pains such evidence is by no means conclusive Embolic phenomena secondary to the endocardial involvement in acute rheumatic fever are rare

**Physical Examination** Detecting the presence of a disease that attacks nearly all the structures of the body containing fibrous tissue calls for a complete physical examination not merely a cardiac study The color of the skin and mucous membranes should be noted Since petechiae nodules and skin rashes are important in diagnosis the patient should be stripped Careful observations should be made before during and after the examination for the presence of twitchings Nose throat ears sinuses teeth and pharynx should be inspected for evidence of infection The lymph nodes draining these areas should be carefully palpated and roentgen examinations made when indicated

CARDIAC EXAMINATION generally reveals little in the early stages In some instances the only signs aside from the tachycardia may be furnished by an electrocardiographic study Here prolongation of the conduction time (pages 615 139) may point to an acute rheumatic carditis Signs of organic valve lesions are not present early in the disease If signs of pericarditis are present usually they have been preceded by more easily recognizable signs of cardiac involvement Enlargement of the heart may

be evident on percussion and it may be accompanied by the systolic apical murmur of relative mitral insufficiency This soft murmur is usually the first on the scene and the question always arises as to its exact significance That it may at times be functional is shown by its disappearance when the acute infection subsides and the reappearance later of a murmur of harsher pitch which is more significant evidence of organic valvular involvement

VALVULAR LESIONS In the later course of the rheumatic disease valvular lesions offer abundant evidence of the presence of a cardiac invasion in fact so absorbed does the student become in their study that other signs are often unrecognized Mitral valve disease is most frequent and the



FIG 55 Rheumatic heart disease Stenosis of the mitral valve viewed from above

harsh systolic apical murmur of uncomplicated regurgitation is more common in children It is attended by an accentuated pulmonic second sound and cardiac enlargement With recurring infection or with increased contraction of the scar tissue in the valve following the initial invasion stenosis develops The first reliable sign of its presence is a mid-diastolic apical murmur To elicit the signs of early mitral stenosis the patient should always be examined in both the erect and recumbent positions Many times a murmur that is inaudible or questionable in the erect position becomes clearly evident in recumbency after exercise particularly if the patient is examined lying on the left side Later in the course of the disease the diastolic murmur may be readily heard in the erect position, and the typical presystolic accentuation will be recognized As the stenosis of the mitral valve advances (Fig 55), the first heart sound at the apex becomes louder and acquires a decided slapping quality Palpation over the area of the apex will now reveal a diastolic thrill Definite alteration in the cardiac silhouette occurs when mitral stenosis becomes established (Figs

20 21) and characteristic changes may also appear in the electrocardiogram (See Fig 190)

In advanced mitral stenosis the left auricle becomes dilated Further weakening of its walls by the inflammatory process that accompanies the rheumatic state makes it a vulnerable spot and sooner or later auricular fibrillation replaces normal rhythm (page 145) When co ordinated auricular contractions cease the crescendo quality of the apical diastolic murmur disappears and only a short murmur is heard in this area in early diastole The diagnosis of stenosis will then depend on the other features of the case, particularly the quality of the first heart sound and the appearance of auricular fibrillation in a patient with a definite rheumatic history

When rheumatic infection attacks the aortic valve the same changes occur and result first in regurgitation and later in stenosis No sign of involvement of the aortic valve appears on physical examination until a cicatricial retraction produces regurgitation As a portion of the blood projected into the aorta during systole flows backward through the damaged valve into the ventricle in diastole it produces a blowing murmur heard best along the left sternal border at the third or fourth interspace The murmur of aortic regurgitation may be heard at the end of expiration with the patient in the upright position and bending forward Faint diastolic murmurs of early aortic regurgitation may sometimes be detected only by the use of the Bowles stethoscope attachment This murmur is transmitted in the direction of the regurgitant stream of blood and may be heard as far down as the cardiac apex The aortic regurgitant murmur is audible in early diastole a fact that at once serves to differentiate it from the late diastolic murmur of mitral stenosis In addition the diastolic murmur of aortic regurgitation is a high pitched blowing murmur while the diastolic murmur of mitral stenosis is low pitched and has a rumbling quality The mitral diastolic murmur is heard over a small area in the region of the apex while the aortic diastolic murmur has a much wider distribution

When the degree of rheumatic involvement becomes greater and the cusps of the aortic valve adhere a systolic murmur appears over this valve area which as the degree of stenosis increases becomes more harsh and more intense A systolic thrill is palpable over the aortic area at this stage and the aortic second sound is diminished or absent A plateau type of pulse and a low pulse pressure can usually be demonstrated

In any rheumatic case the physical signs of aortic or mitral valvular disease depend on the relative degrees of regurgitation or stenosis present If regurgitation is the main lesion at the aortic area the diastolic murmur is long and loud and the peripheral vascular signs striking while if stenosis predominates the systolic murmur over the aortic area will be the main element and the diastolic murmur although heard in the same area will be shorter and less intense When aortic regurgitation is marked there are characteristic alterations in the cardiac size and shape (see Fig

23) Often in patients with aortic insufficiency the degree of left ventricular hypertrophy is extreme

HEALED VALVULAR LESIONS at the mitral and aortic areas are the usual sequelae of rheumatic infection less often involvement of the tricuspid and pulmonary valves occurs Valvular lesions lead to cardiac hypertrophy and in the course of time are followed by heart failure A less frequent manifestation of rheumatic infection that may occur later in life is calcareous aortic stenosis Described first by Monckeberg in 1904 the etiology of this lesion has since formed the topic of much speculation Many clinicians consider it a manifestation of atherosclerosis others claim that it arises as a result of healing of a subacute bacterial endocarditis The majority of observers now consider it as a late manifestation of rheumatic heart disease [413]

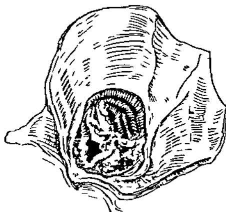


FIG 56 Rheumatic endocarditis The calcific type of aortic stenosis (viewed from above)

IN CALCAREOUS AORTIC STENOSIS fusion of all the cusps is usually seen (Fig 56) The reduced blood supply to this area favors the deposition of calcium and the degree of obstruction becomes extreme Many times these calcium deposits in the valve can be visualized by roentgenologic methods (see Fig 26) Calcareous aortic stenosis is much more frequently seen in males than in females and is accompanied by extreme cardiac hypertrophy It occurs at any age Angina may be present and this complication makes sudden death a possibility

SPECIAL METHODS OF EXAMINATION are valuable aids in the diagnosis of rheumatic infection Aside from the electrocardiographic examination the roentgen ray study (page 37) may be depended upon to reveal characteristic alterations in size and shape of the heart in the presence of all types of rheumatic disease The sedimentation rate is a valuable index of activity (page 57) while the blood count will reveal anemia and leukocytosis The blood culture shows no growth The urinary findings are influenced by the presence of active infection and congestive cardiac failure

## PROGNOSIS

The prognosis of rheumatic heart disease is difficult if not impossible to state when the patient is first examined Caution however must be used in giving too grave an outlook, especially in children in the presence of an active process (page 167) When the infection subsides and the heart balance is in some degree restored a continued although restricted existence, may be possible for many years Conduction defects disappear heart size diminishes even murmurs may fade from the picture along with the *active rheumatic state*[31] *Exceptional cases may recover to such an extent* that on future examination all evidence of the existence of so severe a carditis will be lacking

**Individual Factors** I do not believe that we can predict the outcome of acute rheumatic carditis in any patient by the course that the disease has previously pursued in brother sister or parent While heredity has some bearing on the patient s disease the progress and the ultimate degree of cardiac damage are individual factors and subject to wide variations It can be safely stated however that the earlier the onset the worse the prognosis and the greater the likelihood of cardiac involvement The disease has a tendency to pursue a more fatal course in females than in males It is important to determine and record the number of attacks in the history As a rule the greater the number of attacks the more likelihood there is of a resultant cardiac damage whereas if there is a definite history obtained of a number of rheumatic episodes and the heart shows slight damage it is unlikely that future recurrences will be *any more severe* *As the patient grows older and passes puberty the* physician is less fearful of the cardiac sequelae of the recurring attacks

The discovery of rheumatic nodules (Fig 54) aside from the value in clinching the diagnosis likewise aids in prognosis As a rule the patients showing typical nodules may be expected to have a stormy time and acquire early and usually serious cardiac damage A blond child under 10 who shows fever and nodules will usually cause many an anxious moment before signs of infection become quiescent

**The environment** has a decided influence on the progress of the disease[36] [369] Poor housing conditions dampness and all the aspects of poverty that may be encountered contribute to persistence of the infection, while warmer climates sunshine, and good food have a favorable influence on the course of the disease and consequently are factors that support a more favorable prognosis

**Occupation** plays a decided role in prognosis If strenuous work is carried on by young men and women whose education has been frequently interrupted by episodes of acute rheumatic infection it leads to a greater degree of cardiac disability Those who are fitted for lighter and higher salaried positions survive longer, other things being equal

**Pregnancy** may add to the gravity of the prognosis in some cases (page 452), but it is surprising how well many patients with advanced lesions progress through one or even two pregnancies without adding to the cardiac embarrassment or inducing recurrence of the rheumatic process (Chapter 15) There is an unpredictable (and fortunately rare) danger of invasion of organisms that may attack the already damaged heart valves

**The occurrence of subacute bacterial endocarditis** naturally alters the prognosis at once In this event, instead of remaining a mild case of healed rheumatic infection with a good prognosis the patient becomes a hopeless case This is one of the major tragedies in the course of rheumatic heart disease

**Congestive Heart Failure** If the patient survives the acute attack of rheumatic fever *and is fortunate enough to avoid the complication of* subacute bacterial endocarditis the possibility of congestive heart failure in middle life lies ahead Slight lesions in patients who are carefully watched may be compatible with a long life Willius has shown that the average age at death of the patient with mitral disease is 30 years 32 years when both aortic and mitral valves are involved and 43 years when the aortic valve alone is affected [411]

**Thrombus Formation** In patients with advanced mitral stenosis the late course is very apt to be complicated by auricular enlargement and auricular fibrillation This combination paves the way for another danger for under these circumstances slowing of the blood current in the atrium favors thrombus formation Particles of the clots then become dislodged and form emboli

**Hemoptysis** Increase in the pulmonary pressure that follows advancing stenosis may cause capillary rupture and hemoptysis may appear This symptom in later life again suggests the diagnosis of tuberculosis particularly if the patient shows fever and pallor and has a history of loss of weight However tuberculosis is very unusual in patients with mitral stenosis

## TREATMENT

While it is true that we are still groping in the dark for the cause of rheumatic infection careful management can accomplish much Except for the almost specific value of salicylate therapy in joint involvement we have no potent drug or serum to use in our fight which in most cases results in a long siege However gratifying results are many times possible if we adopt and follow a definite plan of therapy

The first essential to success is to obtain the full co-operation of the patient and his family This is accomplished by acquainting them with the nature of the disease and the basic principles of the method that will be used in combating it The more the patient knows about the type and extent of his cardiac disability the better equipped will he be for the future When the disease remains smoldering after the mildest and most

innocent onset the danger is usually the greatest for many times the patient's consent to the necessary limitations is apt to be obtained with difficulty

## Rest

Although there is a wide variation in the extent of the involvement and consequently in the symptoms presented by each patient the basic rules for management are the same for all The most successful treatment of rheumatic infection pays close attention to the cardiac condition Consequently when a diagnosis of a generalized rheumatic infection is established a rule of treatment should be to take it for granted that an acute myocarditis is present Exertion should therefore be totally restricted in all cases and the patient kept in bed until the signs of activity have faded

**When do we allow an increase in the activity?** This decision is not easy for there is no absolute guide that may be used in all cases The leukocyte count is not trustworthy in every instance The temperature may likewise be misleading since it is possible for activity to be present in many children who will show no temperature elevation The pulse rate while many times a most reliable guide is by no means an infallible one A drop in the pulse rate is a good omen in conjunction with the other signs but per se it may be misleading for bed rest in some patients may produce a satisfactory drop in the pulse rate in spite of a persisting and progressing infection

**The sedimentation rate** I believe is a valuable guide in determining the status of the disease (page 57) It is increased during the active phases and returns slowly to normal when infection subsides However the sedimentation rate is not a specific test for rheumatic infection since all inflammatory processes with or without fever influence it It is wise always to consider the sedimentation rate in the light of the other findings and never to depend upon a single determination as a guide to future treatment Comparative readings should always be made If these remain consistently high active rheumatic infection is present Occasionally the sedimentation rate will fall to normal in the presence of congestive failure, in this instance the fall may be regarded as a bad sign

Body Weight A record of the body weight should always be kept during the period of bed rest since a gain in weight occurs in convalescence, and when accompanied by other favorable signs it permits a cautious increase in activity As a rule the period of bed rest prescribed in each case will depend upon all the clinical evidence The minimum is usually two months, although much longer periods become necessary in severe cases

## Heart Hospital

Bed rest is most satisfactory in a special hospital or convalescent home although it may be possible in some selected instances to accomplish it in the patient's own home Environmental conditions in each case should guide the physician in making this decision



A



B

Fig 57 A The Children Heart Hospital B A school session Children's Heart Hospital

Stroud and McMillan[36] have called attention to the need in this country of increased facilities for prolonged convalescent care of children with active and latent rheumatic carditis More specially equipped hospitals are needed, for they can carry on this work with a higher degree of efficiency than the ordinary hospital Each patient requires proper grading of his exercise and the supervision of workers who are skilled in the detection of signs of early activity *But most important of all heart hospitals* are equipped to carry on the long siege against rheumatic infection and do not neglect the scholastic education of their patients during the many months of bed rest (Fig 57) Education is the great need of the young cardiac patient because adequate training may enable him to obtain a sedentary occupation at a later date

Children are admitted to heart hospitals for long periods usually from 12 to 18 months and only those cases are accepted that may be expected to derive benefit from the course of treatment The requirements for admission to the Children s Heart Hospital in Philadelphia are as follows [360]

1 *Age limit Boys 3 to 1[illegible] years girls 3 to 13 years*
2 Children shall have either possible or potential heart disease
3 No patients who have had congestive cardiac failure or who have a hopeless prognosis shall be admitted
4 Before admission each child shall spend at least two weeks in a hospital
5 As far as possible all foci of infection should be removed
6 Parents shall agree that children will remain in the hospital from three to six months or longer, at the physician s discretion

TABLE IV

DAILY ROUTINE IN CHILDREN S HEART HOSPITAL (Stroud and McMillan)

| | | |
|---|---|---|
| Temperatures | 7 to 7 30 a m | |
| Breakfast | 7 30 a m | |
| Older children attend school from | 9 to 11 30 a m | |
| Younger children rest from | 9 30 to 11 30 a m | |
| Younger children attend school from | 11.30 to 12 30 p m | |
| Older children rest from | 11 30 to 12 30 p m | |
| Dinner | 12 30 p m | radio |
| Temperatures | 1 to 1 30 p m | |
| Younger children attend school from | 1 30 to 3 p m | |
| Older children play until 2 30 and rest from | 2 30 to 4 p m | |
| Younger children rest from | 3 to 4 p m | |
| Play hour | 4 to 5 15 p m | |
| Temperatures | 5 15 p m | |
| Supper | 5.30 p m. | radio |
| Bedtime | 6 15 p m | radio |

*Trans Sec Dis of Children of Amer Med Asso 1937 Reprinted by permission of Amer Med Asso

Table IV gives an idea of the average schedule of the patients in the Children s Heart Hospital in Philadelphia Reviewing the first 225 cases subjected to this special environment and care Stroud and McMillan reported marked improvement in 108 children These patients on discharge were able to carry out practically the same daily routine as healthy children of the same age and social status

During the prolonged convalescent period that follows an attack of

rheumatic infection regular visits to the physician s office should be made The patient should be told that these visits are not for the purpose of obtaining medicine, but for the detection of any clue that might suggest a recurrence of the infection When the physical status is determined at each follow up examination suitable adjustments in the exercise allowance may be made This prescription is based on the extent of the lesion and the weight but is mostly governed by the symptoms if any produced by the previous exercise allowance In the case of children a contact should be established with the school authorities in order that co-operation may be obtained in carrying out the planned program

Some patients visit their physician every month others every two three or even six months as the circumstances demand It can be truthfully stated that advanced cardiac disease develops more often in those who refuse to co-operate with the physician or clinic in the matter of these routine follow up examinations However it seems that it will always remain a problem to convince patients and their parents of the necessity of regular visits to the doctor when they feel fine particularly when no special therapy is carried out at the time of the visit and no prescription for medicine is forthcoming Many times in cases of this type some form of physiotherapy or even a placebo may prove valuable in management After all anything that ultimately proves of advantage to the patient may be viewed as good treatment

### Management of Arthritis

Although I cannot prove my point by an array of statistics I believe that the joint manifestations of rheumatic infection are less often seen today than they were 20 years ago A possible explanation lies in the freer use of proprietary preparations earlier in all fevers by the modern American family

The salicylates have a specific action on the joint manifestations of rheumatic fever When they fail the reason is either insufficient dosage or a mistaken diagnosis Sodium salicylate in doses of 4 0 to 7 0 Gm (60 to 100 grains) daily in small children and higher in older children and adults usually eliminates all joint symptoms within 24 hours The fever likewise decreases as the joint signs disappear (see Fig 59) Salicylates should always be administered with an alkali in the form of an equal amount of bicarbonate of soda The error frequently is made in giving too small rather than too large doses of sodium salicylate If gastric irritability occurs with doses which are inadequate to control the joint symptoms the salicylate can readily be given by bowel A dose of 8 0 Gm (120 grains) mixed in eight ounces of thin starch paste may be given twice daily Only rarely is it necessary to resort to the intravenous use of salicylate but when necessary a 10 cc ampule of a 20 per cent solution can be given two or three times a day Prompt relief of pain follows this method and if the injections are given slowly reactions are few

Administration of salicylates in very large doses will produce in some

patients a toxic state due to acidosis, and it is most important to recognize this if it occurs Vomiting is a common symptom at the onset and may be followed by increased respiratory rate fever, restlessness and coma Salicylism may be confused with diabetic acidosis, but the differentiation is possible by blood sugar determination Tinnitus deafness twitching convulsions and delirium are symptoms more characteristic of salicylate poisoning than diabetes Toxic signs following excess salicylate administration are accompanied by a lowering of the carbon-dioxide level of the blood The symptoms are relieved by alkalis

The joints involved in acute rheumatic fever should be wrapped in cotton and protected from the weight of the bed clothes A 20 per cent methyl salicylate ointment may be used if preferred Since careful nursing care is most essential the acute cases should be sent to the hospital whenever possible

### Management of Chorea

When the rheumatic infection invades the brain and choreiform movements develop the patient should be moved to a peaceful quiet environment Again efficient nursing care is essential If the movements persist sedatives should be used freely Either elixir of phenobarbital 4 0 cc (1 fl dram) sodium bromide 0 6 to 1 0 Gm (10 to 15 grains) or chlorotone 0 3 Gm (5 grains) given after meals for a few days are helpful The salicylate medication should be continued Although the drug has no specific effect on either the nervous system or cardiac involvement it is well to continue its use if the rheumatic invasion is active in localities other than the joints and is attended by pain

**Fever Induction** The most recent and perhaps the most useful form of therapy for rapid relief of the symptoms of chorea is fever induction by means of the Kettering apparatus [307] While it is possible to produce fever by intravenous injections of typhoid bacilli it is best to use the induction method Lately other forms of rheumatic infection have been treated by fever induction and claims made that it shortens the duration of the attacks (page 523)

### Other Forms of Therapy

**Tonsillectomy** Since the belief that the tonsils are the portals of entry of the rheumatic infection is prevalent no time is usually lost in removing them as soon as rheumatic activity is suspected Often this is a grave risk for severe recurrences after tonsil operations are not at all uncommon **Tonsillectomy should be carried out only when the infection has been found to be quiescent It is never an emergency**

While considerable difference of opinion prevails concerning the ultimate value of this procedure it is certainly wise to plan a tonsillectomy in rheumatic patients who show evidence of tonsillar hypertrophy infection or secondary involvement of the cervical glands Some protection against the temporary blood stream invasion that may follow is offered by the

administration of sulfanilamide in the proper dosage before and for a short time following the operation (page 198) While sulfanilamide may be of value in preventing a dangerous bacteremia following tonsillectomy it does not affect the course of established rheumatic infection [61, 69, 370]

**Dietary measures** should be directed toward keeping up the body weight A high caloric high vitamin diet is valuable This may be fortified by the addition of 50 mg of crystalline ascorbic acid (vitamin C) twice daily and 4 0 to 5 0 Gm of brewer s yeast (vitamin B) daily in the form of tablets (Chapter 21)

**Sera and Vaccines** Attempts at a more specific type of therapy have led to the administration of various sera and vaccines The results reported show no uniformity and are all open to question It must be remembered that we do not know the exact nature of the rheumatic infection and until we do such favorable results that follow the injection of the various preparations made up from the streptococcus must be regarded as non specific I have seen the indiscriminate use of vaccines many times light up a previously inactive rheumatic infection Although I do not deny that the introduction of foreign protein will aid the mechanism of defense in some cases I do not feel justified in attempting it particularly when the patient is progressing slowly with the aid of his natural forces The infection that sleepeth harmeth nobody

**Prevention of recurring upper respiratory tract infection** in our rheumatic cases* would be a most efficient means of lessening the number of attacks of rheumatic fever Coburn[69] and others have transported rheu matic children to tropical and subtropical climates and have demonstrated that they are free of upper respiratory infection in these warmer latitudes and that all the rheumatic manifestations subside to return again when they are re exposed to the climatic conditions of the northern seaboard states

**Roentgen ray treatments** over the precordium have been advocated by some workers in the hope of limiting infection and minimizing cardiac damage As far as I can determine these exposures have no effect what soever on the heart and appear to be ineffective and represent purely local thrusts at a generalized disease Levy and Golden have recommended roentgen ray therapy for various forms of rheumatic infection which show a low grade of activity The changes recorded in the electro cardiogram the clinical improvement and *the relief of pain experienced* by some cases suggested to these observers that such exposures have had a beneficial effect They have reported a series of 48 cases seen during the past 11½ years No harmful effects of irradiation were noted The manner in which improvement is accomplished is unknown

**Valvulotomy** The surgical procedure of valvulotomy in mitral stenosis aside from the difficulty in the technic and the risk involved offers little even if successful for the fibrous tissue in the valve may soon re establish

* Utopia in Philadelphia

the stenosis Where marked cardiac hypertrophy is present, rib resection has been occasionally successful

## PREVENTIVE MEASURES

Early recognition of all the manifestations of the rheumatic state from growing pains to frank articular involvement and prompt treatment may aid in preventing advanced cardiac change Where carditis is the only sign of the presence of the disease, frequent physical examinations of all school children make possible the early recognition of rheumatic infection Suspicion of its presence should be entertained in all children who show pallor anemia loss of weight and who complain of weakness or frequent unexplained nosebleeds By the discovery of early cardiac involvement we may in some measure prevent advanced hopeless lesions

As far as the actual prevention of the disease itself is concerned further investigations are needed by both clinician and laboratory worker As Coombs[7] has stated

> Life is short—but art is long and it is useless to attempt the annihilation of such a disease as this by an 'intensive campaign — To the conquest of disease there can be no short cuts It is a painful hand to hand struggle in which everyone must be mobilized and in no part of the battle front is there better opportunity for successful co operation between all sections of the profession—general practitioner consultant and administration—than in that on which we are faced by rheumatic heart disease

## ILLUSTRATIVE CASES

### Suspected Rheumatic Heart Disease—The Evaluation of the Systolic Murmur When It Represents the Only Abnormal Finding

**Case 9** *P F male aged 10 years was sent to the Cardiac Clinic of the Memorial Ho p al on May . 1936 by a school physician Complaints were nervousness and slight weight loss*

History The birth had been normal There was no history of rheumatic infection He had a tonsillectomy at the age of five There were no subjective symptoms referable to the cardiovascular system

Physical Examination Well developed lad Throat clean Entire examination normal except for the presence of a soft, systolic apical murmur poorly transmitted into the axilla The apex beat was in the fifth intercostal space well inside the mid clavicular line The heart sounds were normal B P 110/80

*Laboratory Data Wassermann reaction negative Blood count normal* Sedimentation rate 5 mm in one hour (Fig 58C) A roentgen study (Fig 58A) showed no cardiac enlargement The electrocardiogram (Fig 58B) was normal

Clinical Diagnosis Class E Signs referable to the heart but the diagnosis of heart disease cannot be proved Murmur functional in type Activity unrestricted

**Discussion** Well established lesions at the mitral valve accompanied by cardiac enlargement usually offer no difficulty in diagnosis However a systolic murmur heard at the apex in otherwise healthy children continues

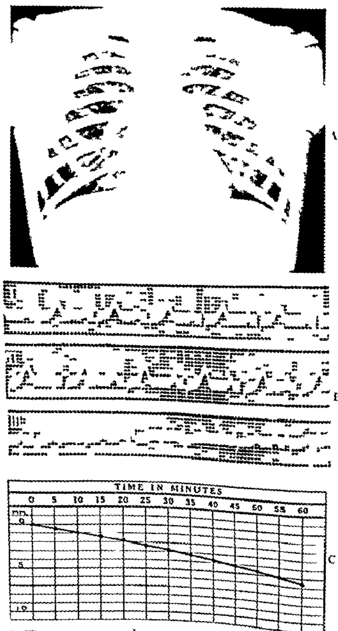



FIG 58 A The roentgenogram shows no cardiac enlargement
B The electrocardiogram is within normal limits The notching and low voltage of QRS3 not infrequently occur in the tracings of normal patients
C Sedimentation chart The red blood cells show a fall of 6 mm in one hour

to be a stumbling block in cardiac diagnosis Although I admit that in some cases the correct interpretation at the time the child is first seen is difficult or impossible the evaluation of the importance of the finding in the majority of cases presents very little difficulty if certain fundamental facts are kept in mind

In the first place many healthy children may show a transient soft apical systolic murmur following excitement or exercise when the speed of the circulation is increased It may likewise accompany fever or anemia The murmur in this region may be heard only during a certain phase of respiration or it may appear when the patient is in recumbency, disappearing in the erect position The terms 'functional' "hemic' and cardiorespiratory ' are used in describing the systolic murmurs accompanying these conditions Although these are acceptable terms care should be used in describing the intensity pitch quality and duration of the murmurs heard (page 19) If this is done in all cases, the data obtained aids in the correct evaluation of the finding For example functional murmurs are more apt to be faint or moderate in intensity, while loud murmurs are nearly always associated with organic heart disease Harmless murmurs are more apt to be short and blowing while those of long duration particularly if they are harsh rumbling or crescendo, generally accompany disease of the valvular structures

This boy had a faint blowing systolic murmur over the mitral area Its duration was short and it increased to moderate intensity in the recumbent position Examination showed the absence of cardiac enlargement So far the criteria for a functional murmur have been fulfilled However the findings must be viewed in the light of the patient s past history Did he have rheumatic fever or chorea? All we can elicit is a history of nervousness It is unlikely that this symptom is related to chorea, since the blood count sedimentation rate temperature and weight furnish no supporting clues

If the patient had given us a clear cut history of rheumatic fever the evaluation of the murmur would have been more difficult In these cases, if no cardiac enlargement accompanies the systolic murmur and in the presence of negative laboratory studies a definite answer cannot be given at once Re examination should be made two or three times a year Meantime since we are aware of the frequency of valvular disease following rheumatic fever full activity should not be allowed Certainly competitive sports should be eliminated until the child is older If as time goes by and puberty is passed the findings remain constant we can be certain that the heart has escaped and full activity may be permitted On the other hand if the murmur becomes louder and longer and cardiac enlargement appears accompanied by an increased pulmonic second sound a mitral regurgitation is probably present

Sometimes the systolic murmur in the mitral area disappears This is possible following the correction of severe anemia (page 424) or hyperthyroidism (page 356) A rapid heart rate following exercise may be accom

panied by a systolic murmur that disappears following rest. In these instances the increased velocity of the blood accounts for the production of the murmur.

### Rheumatic Heart Disease—Acute Carditis Following Typical Joint Involvement—Mild Course

**Case 10.** Mrs. C. Z., a housewife of 7, was well until two days before admission to the Woman's College Hospital on December 7, 1938. She first noticed sore throat which was followed a week later by high fever, s eating and pain and swelling in the left knee. There was no history of previous similar attacks.

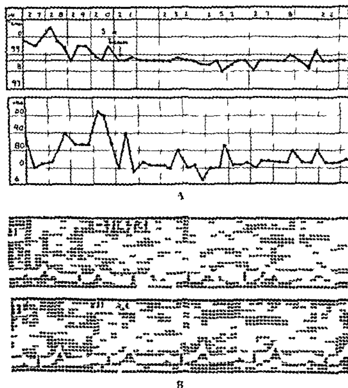


Fig. 59. A. Temperature pulse record.

B. The electrocardiogram. In the first record (lead ) taken on 1/14/38 note prolongation of the P-R Intervals o 0.3 econd. In the second record (same lead) taken on 1/10/39 the P-R Intervals are normal.

Physical Examination. B.P. 110/80. T. 00. F. Pulse 86. Respirations 20. Well nourished adult fem l not acutely ill. Tonsils red and enlarged. The left knee was swollen and tender with restricted motion. The heart was normal in size. The apex beat was palpable in the fifth interspace ll in de the midclavicular line. A soft systolic murmur was heard in the mitral area. The rhythm was regular.

Laboratory Data. W.B.C. 13,000. R.B.C. 4,500,000. Hemoglobin 84 per cent (Sahli). The electrocardiogram showed prolongation of the P-R intervals to 0.34 second (Fig. 59B).

CLINICAL DIAGNOSIS A Etiologic Rheumatic Active B Anatomic No cardiac enlargement Mitral regurgitation?? C Physiologic First stage heart block (Prolonged P R intervals) D Functional Classification Class I Therapeutic Classification Class E

Discussion The onset here is quite typical An attack of tonsillitis was followed by pain and swelling in the left knee It would be hard to ignore such evidence The large infected tonsils that were seen in this patient withstood many attacks but the rheumatic invasion finally took place In younger patients who have had one or more attacks of rheumatic fever, upper respiratory infections of any type are capable of producing fresh invasions The common head cold must be viewed as a serious complication in a rheumatic subject Each cold threatens to increase the amount of cardiac damage should it succeed in lighting up a latent infection Consequently the rheumatic patient should always go to bed for a few days when the first signs of a head cold appear Unnecessary contacts with individuals who have colds should be avoided as much as possible The rheumatic subject should also be carefully examined for signs of activity when the symptoms of respiratory invasion have subsided

Bed rest and salicylates were prescribed on admission in this case The specific action of salicylates on the joint symptoms is readily seen in the temperature record (Fig 59A) Four days after admission definite evidence of an acute inflammation of the heart appeared in the electrocardiogram (Fig 59B) This finding was valuable in planning the subsequent manage ment Since the patient felt so much improved after the salicylate therapy, it was difficult to persuade her to remain in bed She was a farmer s wife always used to hard work and had three children waiting at home When the situation was explained the patient consented to remain in the hospital for a longer period of supervised bed rest Salicylates were dropped at the end of the second week and since there was no recurrence of fever or joint pain they were not used again The salicylates could have had no influence whatsoever on the course of the carditis

*During the next eight weeks of her stay in the hospital* no other drugs were used The patient was placed on a high caloric diet and cevitamic acid (vitamin C) was given in 20 mg doses t i d The tonsils although large and infected were allowed to remain They will not be removed until full proof is at hand that the rheumatic infection is quiescent

When the patient was discharged from the hospital to continue her *convalescence at home, the following program was written out and given* to her

First week Sit up in chair beside the bed one hour in the afternoon
Second week Sit up in chair for two hours every afternoon
Third week Sit up in chair one hour in the morning and two hours in the afternoon May walk to the bathroom on the same floor
Fourth week Sit up in chair for two hours in the morning and two hours in the afternoon Walk to the bathroom on the same floor
Fifth week Same
Sixth week Up from 10 A M to 5 P M Do not go downstairs

Seventh week Up from 10 A M to 5 P M May go downstairs at noon and remain there until after dinner
Eighth to tenth week Same
Eleventh week Downstairs for meals One half hour walk out-of-doors in the afternoon
Eleventh to fifteenth week Same but increase walk to one hour by the end of the 15th week
Notes Keep a temperature chart for the first eight weeks Full diet Continue tablets of cevitamic acid (60 mg daily)

Viewing the soft systolic murmur that was heard over the mitral area of this patient on admission in the light of the typical findings we cannot say that it is functional even in the absence of cardiac enlargement During the early days of the infection when a high fever was present the murmur may have been functional in the sense that it was produced in a toxic myocardium by relaxation of the mitral ring Actual inflammatory changes may have already developed in the valve distorting its leaflets and directly interfering with its function When these murmurs persist during the months following convalescence it is reasonable to suppose that they are caused by disease of the mitral valve itself Mitral regurgitation should then be the tentative diagnosis especially if cardiac enlargement can be demonstrated It is true that the diagnosis of mitral regurgitation is very rarely upheld at necropsy However it is equally true that patients do not succumb during this stage of the disease Survival permits contraction and healing of the valve leaflets to take place and mitral stenosis develops Consequently if signs of stenosis are discovered in a patient suffering from acute rheumatic fever, previous attacks should be suspected

## Rheumatic Heart Disease with Aortic Regurgitation No Signs of Active Infection and Slight Cardiac Enlargement

**Case 11** F N a 16 year old school boy was sent to the cardiac clinic at the Woman s College Hospital in January 1931 A heart murmur had been discovered by the school physician

History Negative for rheumatism All previous physical examinations were negative No symptoms were present referable to the cardiovascular system The patient had always been active in school athletics and the exercise tolerance was good

Physical Examination B P 130/60 Apex beat in the midclavicular line 8 5 cm to the left of the midsternal line in the fifth interspace The pulse was 80 and of the Corrigan type and the rhythm was regular There was a blowing diastolic murmur along the left sternal border

Laboratory Data Wassermann negative Blood count normal One urinalysis negative Sedimentation rate normal The orthodiagram (Fig 60A) showed slight cardiac enlargement The cardiothoracic ratio was 0 46 the transverse cardiac diameter was 12 9 cm (predicted 11 3 cm) and the cardiac area was 129 sq cm (predicted 113 sq cm) The electrocardiogram (Fig 60B) showed slight flattening of $T_1$ There was no axis deviation

Clinical Diagnosis A Etiologic Rheumatic Inactive B Anatomic Slight cardiac enlargement Aortic regurgitation C Physiologic Normal sinus rhythm D Functional Classification Class 1 Therapeutic Classification Class B

Discussion Watching this lad over the course of the nine years that he has been visiting our clinic has been most instructive When he first came to us during the winter of 1931 he presented the typical picture of aortic

regurgitation No signs of mitral disease were elicited and to confuse the issue further he gave no history of rheumatic infection

The initial study showed the absence of cardiac enlargement, a flat $T_1$ in the electrocardiogram a negative Wassermann and a very good exercise tolerance Since the majority of patients who have organic heart disease at this age belong to the rheumatic group he was tentatively placed in this category and tests were made to rule out the presence of activity Again blood studies including a leukocyte count and sedimentation rate, were normal He was gaining weight and aside from his cardiac lesion appeared to be in excellent physical condition He was advised to discontinue basket ball but no other restrictions were imposed

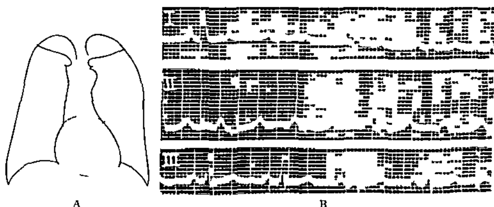



FIG 60 A The orthodiagram shows slight cardiac enlargement Note prominent aortic knob

B The electrocardiogram There is slight flattening of T1 The P waves are widened in lead There is no axis deviation

Although our suggestions were carried out by the school authorities, the patient admitted at each visit that he was participating in competitive sports of all kinds There was no dyspnea As the years went by we felt less concern when we noted that the heart size and other features of the examination remained exactly the same as initially recorded

For eight years this boy has enjoyed unrestricted activity and has taken part in competitive sports against advice However our protests in recent years have been milder, when we observed the absence of any demonstrable ill effect The exercise that we prohibited appears to have added to his mental and physical well being At the age of 24 his chief dangers appear to be the chance development of subacute bacterial endocarditis on the previously damaged heart valve and the recurrence of the rheumatic infection As he grows older the latter danger becomes much less although unfortunately the former possibility remains

During the time this patient has been under our care no routine medication of any kind has been prescribed A tonsillectomy was performed

during the first summer, and he has had a routine dental examination every six months

## Rheumatic Heart Disease—Active Infection—Death from Congestive Failure at Unusually Early Age—Autopsy

**Case 12** E W a male infant of 3½ years was admitted to the Philadelphia General Hospital on 6/7/37 and died on 6/11/37 Chief complaints on admission were fever and frequent nose bleeds

History Bronchopneumonia at ?½ months whooping cough at one year and measles at the age of two Two months prior to admission the patient began to have fever at night and there was a steady weight loss Frequent severe nose bleeds followed and continued until the day of admission

Physical Examination showed an acutely ill child pale slightly cyanotic and poorly nourished T 101 P 1 0 R 35 Pale conjunctivae teeth carious The tonsils were large and cryptic The lungs were clear The heart was enlarged to the right and left The apex was ell beyond the mid lavicular line where a diastolic thrill was palpable A blowing systolic murmur and a rumbling diastolic murmur were heard in the mitral area A to and fro murmur and a systolic thrill were present over the aortic area The liver was palpable four fingers below the costal margin The fingers and toes were short and stubby Rheumatic nodules were palpable n the right knee and the left ankle

Laboratory Data Urine negative Blood hemoglobin 49 per cent (Sahli) R B C 700 000 W B C 9 000 Sedimentation rate 8 mm in one hour The roentgenogram showed cardiac enlargement in all diameters

Clinical Diagnosis A Etiologic Rheumatic Active B Anatomic Cardiac enlargement Aortic stenosis and insufficiency Mitral stenosis and insufficiency C Physiologic Normal sinus rhythm Congestive failure D Functional Classification Class 4 Therapeutic Classification Class E

Discussion Evidence of an active rheumatic infection was present on the first examination In addition to the signs of an unusual degree of cardiac involvement in one so young the cyanosis dyspnea and large liver pointed to congestive failure

In the presence of acute carditis the mechanical strain of the valvular lesions has a much less important place Congestive failure when it occurs in children is usually in itself evidence of activity and if carefully searched for the signs will be brought to light In this child rheumatic nodule were palpable on admission

In cases where the overwhelming infection precipitates cardiac failure digitalis is of doubtful value Nevertheless it should always be given (page 84)

Since cyanosis was marked the child was placed in an oxygen tent which seemed to have little influence on the course of the disease The respiratory rate mounted and the congestive manifestations increased One half cc of mercupurin in 10 cc of saline was given intravenously but had little effect Death occurred on the fourth hospital day

Autopsy The heart (Fig 61 A and B) weighed 150 Gm The epicardium was gray and glistening The myocardium was moderately flabby and contained numerous delicate gray streaks and pin point bright pink areas especially near the endocardial surface The auricular endocardial surface was thick and opaque The free edges of the mitral valve were thickened and scarred The mitral chordae were thickened shortened and adherent The aortic orifice was narrowed just admitting a small probe The aortic

leaflets were thickened rigid and firmly glued together The valves on the right side of the heart appeared normal The foramen ovale was closed The coronary vessels were normal




FIG 61 Rheumatic heart disease There is a characteristic shortening and fusion of the aortic leaflets resulting in stenosis and insufficiency (Autopsy No 33 574 Philadelphia General Hospital )

## RHEUMATIC HEART DISEASE WITH MITRAL STENOSIS AND CONGESTIVE FAILURE—INFLUENCE OF MULTIPLE PULMONARY EMBOLI ON COURSE AND PROGNOSIS

**Case 13** Mrs A H a white housewife of 5 was admitted to the Philadelphia General Hospital on /13/34 complaining of cough shortness of breath and swelling of the legs

HISTORY Increasing dyspnea during the past year A week before admission edema of the legs developed and was followed by chest pain cough and hemoptysis Her mother had Tb and heart trouble No history of rheumatic infection The patient considered herself in good health until a year before admission

PHYSICAL EXAMINATION P 120 (irregular) BP 145/90 Jaundice was present The neck veins were distended There were rales at both lung bases There was edema of the legs to the hips

The heart was enlarged LB 13 0 cm to the left of the midsternal line There was a short diastolic murmur over the region of the apex beat The liver edge was distinctly palpable 7 cm below the costal margin in spite of distention and shifting dulness in the abdomen (Fig 6 A)

The electrocardiogram showed auricular fibrillation with a rapid ventricular rat (Fig 6 B)

CLINICAL DIAGNOSIS A Etiologic Rheumatic Inactive B Anatomic Cardiac hypertrophy Mitral stenosis and insufficiency C Physiologic Auricular fibrillation Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

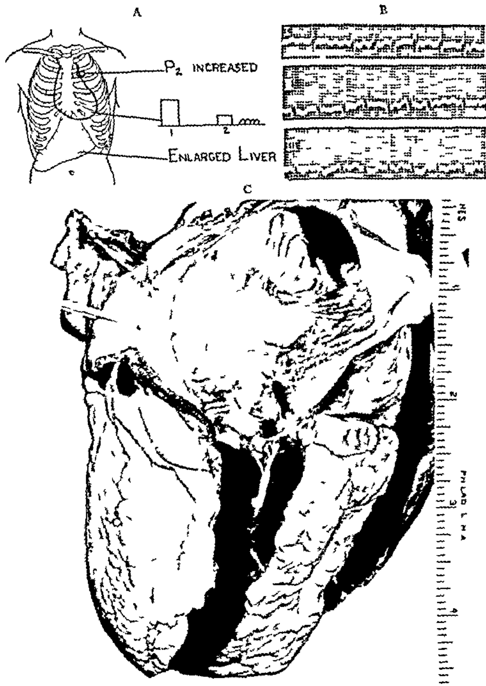



Fig 6 A Cha t of physical findings B The electrocard ogra n Au icular fibr l lation is present v th a rap d ventr cular rate
C Rheumatic heart disease Mitral stenosis s present with marked dilatation of the left auricle Note that the s ze of the aur cle is nearly equal to that of th ventricle (Autopsy No 27 083 Philadelphia General Hospital )

COURSE Down hill from time of entry The patient showed no response to digitalis and diuretics and died on the ninth hospital day

AUTOPSY (Fig 6 C) Rheumatic heart disease with mitral stenosis and cardiac hypertrophy The auricles were dilated There were multiple pulmonary infarcts Pulmonary atherosclerosis Congestive cardiac failure

**Discussion** The diagnosis of the type of heart disease present in this patient was not difficult even in the absence of a definite rheumatic history Symptoms began a year before admission at the age of 51 showing that advanced mitral disease may at times be present for many years and cause no disability Dyspnea was first noticed This increased gradually over the course of a year and finally a week before admission edema appeared and progressed so rapidly that she was forced to enter the hospital

Since this was the first spell of congestive failure in a patient who had rheumatic heart disease a form that is usually characterized by the ability to weather many episodes of decompensation we felt certain that circulatory balance would be quickly restored following the use of digitalis in proper dosage with suitable diuretics The combination of heart failure and auricular fibrillation with a high ventricular rate led us to adopt an attitude of optimism that future events proved was unjustified Although the history of pulmonary infarction was clear cut we failed to consider the unfavorable effect that this complication of advanced mitral disease has on the outcome in the presence of congestive failure

A proper evaluation of the symptom of hemoptysis during the course of mitral disease is important if these errors in prognosis are to be avoided Sudden episodes of chest pain dyspnea and hemoptysis do not always accompany a pulmonary infarction However dyspnea that becomes progressively worse followed by hemoptysis and the signs of congestive failure and shows no improvement when proper treatment is instituted should always suggest the presence of a pulmonary infarct

McGinn and White [17] in an analysis of the incidence of pulmonary infarction in 52 cases of mitral stenosis in 2500 consecutive necropsies showed that this complication occurred in 61 per cent of 23 cases where congestive failure appeared and in only seven per cent of the cases when congestive failure was absent In contrast to this group a study of 82 cases of hypertension showed 39 cases of congestive failure in 21 per cent of which there was pulmonary infarction

When symptoms suddenly become aggravated as in this case and are followed by collapse and increase in the signs of cardiac failure, pulmonary infarction should be suspected (Fig 63) Hemoptysis and chest pain may or may not be present If the patient survives a local pleural friction rub often appears and is followed by fever and leukocytosis A few days later jaundice may be noticed which is of the hemolytic variety and follows the extra burden placed on the already congested liver by the blood pigment from the pulmonary infarct In this patient an icterus index of 20 was obtained a few days after admission

The pulmonary infarction that so commonly complicates advanced

mitral disease accentuating the signs of congestive failure and rendering the patient so refractory to treatment does not always follow embolism Quite often conditions in the pulmonary circuit predispose to thrombosis There is usually present a sclerosis of the lung vessels and when a slowing of the blood current through them occurs during the early stages of heart failure spontaneous clotting is favored An infarct then develops its size

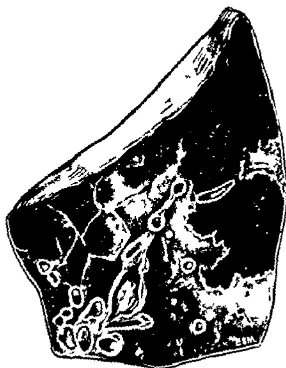


Fig 63 Hemorrhagic infarct of the lung

depending on the caliber of the vessel obstructed At times the discovery of dulness over these areas of infarction in a patient who shows fever and other signs of an active rheumatic process leads to a diagnosis of rheumatic pneumonia Again in cases where the onset is sudden and the area of infarction large the diagnosis of a lobar pneumonia may be made especially if chest pain a temperature elevation hemoptysis and a pleural friction rub appear

The course of rheumatic heart disease is often complicated by embolism after the onset of auricular fibrillation at which time considerable hypertrophy of the atrial musculature is generally present Under these circumstances the detachment of a part of a thrombus and its lodgment in the

lung is not unusual *Pulmonary embolism may also follow thrombosis in* one of the large pelvic or thigh veins although the exact site of origin may be unrecognizable clinically

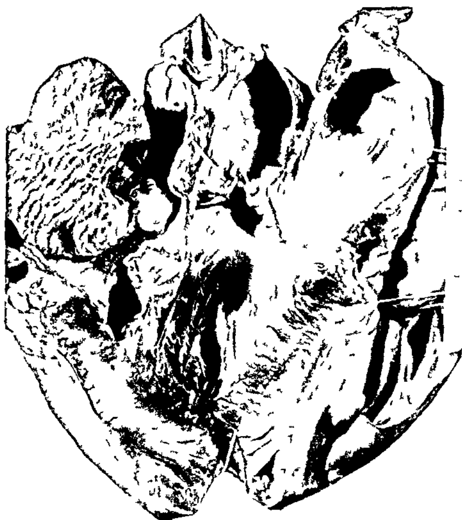


Fig 64 Rheumatic heart disease Note cardiac hypertrophy aortic and mitral stenosis The aortic valve (marked by arrow) is viewed from the sid Its leaflets are fused and thickened A large thrombus with a rippled surface almost completely fills the left auricle The mitral orifice (seen below the thrombus) is sclerosed and contracted (Autopsy No 30 167 Philadelphia General Hosp tal)

The auricular appendages are the common locations for thrombus formation In some cases we may be unable to prove the point of origin of the embolus if no clot is found at autopsy, while in others the whole auricle may be filled with thrombus (Fig 64) Occasionally following coronary

thrombosis mural thrombi may form in the left ventricle (see Fig 109) These particles may become detached and find their way into the systemic or cerebral circulation with serious consequences Ulcerative plaques in patients with advanced arteriosclerosis are often the sites of formation of thrombi that may eventually become dislodged Aneurysms or injured vessels likewise may be the starting points of emboli In chronic valvular disease it is rare for emboli to arise from the vegetations on the heart valves while the auricular source is quite common On the other hand in acute and subacute bacterial endocarditis fragments frequently become detached from the valves and since the mitral and aortic valves are involved most often the embolic signs appear in the systemic circulation

## Rheumatic Heart Disease—Mitral Stenosis—Embolic Manifestations

**Case 14** M M a 30 year-old white clerk was first seen in July 1936 complaining of cough and shortness of breath He had been well until two days before when he developed a severe head cold This was followed by fever and cough and he noticed for the first time that his feet were swollen and that his pulse was rapid and irregular He gave a history of two attacks of chorea the first at the age of seven and the second at the age of 13

Physical Examination Temperature 100 F There was a totally irregular pulse of 120 and the respiratory rate was 3 The blood pressure was 1 0/80 Scattered rales were present in both lung bases There was pitting edema of both ankles The heart was slightly enlarged to the left The first sound was accentuated in the region of the apex and a mid diastolic murmur was heard in the same area P was accentuated

Clinical Diagnosis A Etiologic Rheumatic (Chorea) Inactive ( ) B Anatomic Cardiac enlargement Mitral stenosis and regurgitation C Physiologic Auricular fibrillation Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

Discussion The patient was placed at bed rest at home and complete digitalization was accomplished in three days (page 82) Codeine sulfate 0 03 Gm (½ grain) and ammonium chloride 1 0 Gm (15 grains) were given every four hours On the second day 1 cc of mercupurin was given intravenously These measures were sufficient to dispel all signs of congestive failure and to lower the pulse rate to 75 (see Fig 247B) On the fourth day the digitalis was decreased to 0 1 Gm (1½ grains) of the whole leaf daily At the beginning of the third week the patient was allowed to return to work on a restricted program

During the course of the next three years he was examined every three months The auricular fibrillation continued but no signs of congestive failure were discovered at any time His exercise tolerance was fair

In August 1939 following another upper respiratory infection the patient again developed cough but on this occasion there was hemoptysis The pulse rate rose to 130 and digitalis appeared to be ineffective in controlling the fibrillation Bed rest and mercurial diuretics however were finally successful in restoring the balance In five weeks the congestive failure disappeared and the dyspnea improved Digitalis 0 1 Gm (1½

grains) daily and ammonium chloride 1 0 Gm (15 grains) three times daily were continued

One month later, while at work he experienced a sudden pain in the left leg accompanied by numbness and coldness He was removed to the hospital at once At an emergency operation removal of an embolus from the left femoral artery at the site of origin of the profunda was successfully accomplished However the patient died suddenly the next day, following a cerebral embolism An autopsy was not obtainable

This patient presents the picture of chronic rheumatic heart disease with mitral stenosis The infection occurred early in childhood but no symptoms were present for over 17 years Death occurred three years after the onset of symptoms

The first attack of congestive failure appeared rather suddenly following an acute upper respiratory infection, which is not at all unusual Infection of this type is a much more common cause of a break in compensation than overwork although there is a widespread belief that the opposite is true There was no relighting of the rheumatic process inasmuch as compensation was quickly restored and the temperature returned to normal in a few days

Digitalis was begun when congestive failure first appeared and was continued in a maintenance dose to keep the ventricular rate between 70 and 75 until the patient s death three years later In his case the amount needed was 0 1 Gm (1½ grains) daily

Many cases of chronic rheumatic heart disease pursue a similar slow course over many years with occasional breaks in compensation that respond quickly to bed rest and intensive treatment Such a course is possible but is less often observed in cases of arteriosclerotic and hypertensive heart disease while it is quite unusual in cases of syphilitic heart disease

This patient s final illness was again ushered in by respiratory symptoms However this time he had hemoptysis and although there was nothing on physical examination to prove pulmonary infarction the history suggested this possibility Pulmonary infarction also accounted for the poor response to therapy on this occasion and changed the prognosis since we were prepared to expect recurrence of the accident in the same or other locations A month later an embolus from the dilated left auricle found its way into the left femoral artery Emergency surgical measures were successful but a day later cerebral embolism caused the patient s death

The patient whose heart is shown in Fig 64 was a white male of 33 who died following a clinical course quite similar to that of the patient just described The heart here is enormously enlarged with mitral aortic and tricuspid valve involvement the mitral and aortic valves showing an advanced stenosis The greatly dilated left auricle is completely filled by a large ball valve thrombus This complication of the rheumatic valvular disease was the direct cause of death that occurred suddenly six weeks after admission to the hospital

A similar happening is shown in Fig 65 This patient was a white female

of 26 who had suffered from rheumatic heart disease since the age of seven During the last eight years of her life breaks in compensation occurred associated with recurrences of the rheumatic infection She showed excellent response to the usual therapy and was able to carry on her work until the



Fig 65 Rheumatic heart disease with mitral stenosis A massive thrombus occupies the whole of the dilated left auricle (Autopsy No 33 699 Philadelphia General Hospital)

day she was admitted to the Philadelphia General Hospital Death occurred suddenly and unexpectedly seven hours after admission and autopsy showed advanced mitral stenosis and a greatly distended left auricle entirely filled with thrombus which was white in the center and reddened at the periphery There were concentric laminations The entire thrombus meas

ured 5 × 6 × 8 cm Rarely these masses of clot may undergo complete organization with the formation of either pedunculated or ball valve thrombi (Fig 66) Occasionally these smooth loose bodies are discovered at postmortem in the auricular chamber always larger than the stenosed mitral orifice which they may at times acutely obstruct [9] In some patients this intermittent obstruction may give rise to characteristic symptoms that arouse suspicion of the presence of a ball valve thrombus Changes in the circulation in both upper and lower extremities may take place and progress rapidly to the formation of small areas of gangrene in the fingers and toes Full restoration of the circulation in these areas may occur suddenly if the pressure of the ball thrombus is released in which event the color and temperature of the arms and legs quickly return to normal Syncopal attacks may appear when a ball valve thrombus occludes the valvular orifice and sudden death is always a possibility

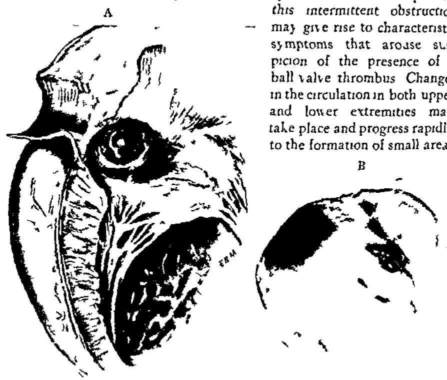



Fig 66 A Ball thrombus in left auricle
B Ball valve thrombus

RHEUMATIC HEART DISEASE OF LONG DURATION COMPLICATED BY AURICULAR FIBRILLATION PULMONARY EMBOLI AND ATTACKS OF PAROXYSMAL DYSPNEA

**Case 15** Mrs M J an American housewife of 51 was first seen in December 1937 complaining of palpitation and dyspnea

HISTORY The patient was well until two years prior to the first examination when dyspnea appeared on exertion About the same time she noticed palpitation and slight ankle edema in the evening There had been a 40 pound weight loss since the onset of illness At the age of 10 she had her first attack of rheumatic fever and was confined to bed for two months

PHYSICAL EXAMINATION B P 150/80 Puls rate 100 Rhythm totally irregular Slight cyanosis and mod rate ankle edema

Heart marked enlargement by percussion both to the right and to the left The apex impulse was palpable in the sixth interspace in the anterior axillary line There was a blowing systolic murmur and a mid diastolic rumbling murmur heard over the apex A systolic murmur was heard over the aortic area

LABORATORY DATA Blood count hemoglobin 82 per cent (Sahli) R B C 3 800 000 W B C 8 000 Wassermann negative

The electrocardiogram showed auricular fibrillation (Fig 67B) The roentgenogram showed cardiac enlargement in all diameters (Fig 67A)

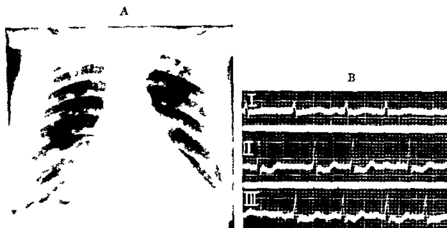



FIG 67 A Roentgen film of chest There is cardiac enlargement with mitralization Note the congestion of the pulmonary vessels and multiple pulmonary infarcts B Auricular fibrillation The ventricular rate is well controlled by digitalis The action of this drug is also evident in the depression of the S T Intervals in leads 2 and 3

DISCUSSION The weight loss described by this patient was evident on physical examination Not infrequently patients with a long standing cardiac lesion will show loss of weight following anorexia that attends congestion in the portal system In others, the poor blood supply may interfere with proper nutrition of essential structures and wasting gradually appears (cardiac cachexia)

Treatment of this patient was begun by two weeks bed rest During this time she was digitalized and the ventricular rate reduced to 70 She was then allowed to be out of bed and about the house The dyspnea improved and the edema did not recur The dyspnea always returned however when further activity was permitted but on a markedly restricted program and maintenance digitalis she was kept fairly comfortable for a year

In December 1938 mild spells of paroxysmal dyspnea appeared following excitement or more than the usual amount of exercise McGinn and White [47] attribute these spells of pulmonary congestion that appear in patients with mitral stenosis to the strength of the right ventricle which in

certain situations pumps more blood into the lungs than can be passed through the mitral valve in time to prevent a stasis Evidence of the congested state of the lungs is furnished on these occasions by the appearance of rales and frothy bloody sputum If the right ventricle does not bear up under the increased load congestion often follows in the portal system with cyanosis large liver, and edema

*These paroxysms became more frequent and distressing during the next* few months The more severe attacks were controlled by injections of morphine sulfate 0 015 Gm (¼ grain) Finally during one of the seizures the patient developed a sharp chest pain that was followed by hemoptysis Dyspnea and other signs of congestive failure appeared on the scene very promptly No doubt the slowing of the blood current in the lungs produced by the delay in emptying favored thrombosis The resulting infarct was a large one judging by the extent of the physical signs that were evident a *few days later in the left lower chest During the next six weeks there were* two additional attacks of pulmonary thrombosis similar to the one just *described Each occurred as the patient was showing some improvement in* breathing and other signs of congestive failure

Following the last attack injections of mercupurin were given every fifth day regardless of the presence of visible edema Ammonium chloride was started at the same time and continued in 1 0 Gm (15 grains) doses after meals Improvement now was gradual but was maintained and the patient *managed to regain her former level of exercise tolerance Following this* restricted program and continuing the digitalis and diuretic maintenance dosage the patient has had no recurrence of her pulmonary symptoms for over a year and a half

## Rheumatic Heart Disease with Mitral Stenosis—the Influence of Hypertension on the Course of the Disease

**Case 16** Mrs F W a housewife of 55 was first seen in June 1937 at which time she complained of shortness of breath palpitation and edema These symptoms had all been present and increasing in severity for four months The patient had two attacks of rheumatic fever in childhood In 1930 a subtotal thyroidectomy was performed because of palpitation nervousness and loss of weight

Physical Examination Blood pressure 60/110 Dyspnea cyanosis and edema of both legs to the knees were present and there was marked distention of the jugular veins

Heart totally irregular rhythm rate 1 0 The left base measured 13 5 cm and the right 4 0 cm There was an accentuation of the first sound over the apex and a mid diastolic murmur There was a systolic murmur as well as an accentuation of the second sound over the aortic area There were no thrills palpable The liver was enlarged *to the umbilicus Rales were present in both lung bases*

Laboratory Data Wassermann negative Urine showed a trace of albumin and a fixed specific gravity of 1 010 on several occasions Blood count normal Basal metabolic rate plus 7 per cent

The electrocardiogram showed on first examination a rapid auricular fibrillation (Fig 68B) Digitalis reduced the rate and inverted the S-T intervals

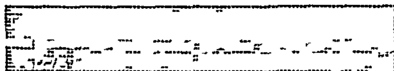
The roentgenogram (Fig 68A) showed enlargement of all cardiac diameters

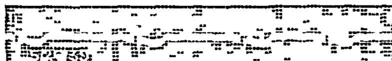
Clinical Diagnosis A Etiologic Rheumatic (Inactive) Hypertension B Anatomic Cardiac hypertrophy Mitral stenosis Mitral regurgitation C Physiologic Auricular

fibrillation Congestive cardiac failure D Functional Classification Class 3 Therapeutic Classification Class E

A







B

Fig 68 A Roentgen film Note marked cardiac enlargement all diameters
B The first strip (lead 1) taken 6/27/37 shows presence of auricular fibrillation The second strip (same lead) taken 5/26/39 shows a marked slowing of the ventricular rate to 58 with inversion of S-T intervals

Discussion The course of the rheumatic heart disease in this patient was complicated first of all by the appearance of thyrotoxicosis at the age of 48 She developed palpitation and dyspnea a slight enlargement of the thyroid exophthalmos tremor nervousness and loss of weight The basal metabolic rate was plus 45

Following a subtotal thyroidectomy the cardiac symptoms were much improved and her exercise tolerance increased During the next seven

years there was a gain in weight of 25 pounds In 1936 before the cardiac symptoms reappeared, the patient was told by her physician that she had high blood pressure

In 1937, her blood pressure measured 260/110 and mild congestive failure was present She was digitalized and proper maintenance dosage continued and in addition she was given elixir of phenobarbital, 4 cc (one dram) after meals A program regulating her daily activities which included a part time maid in the household, a two hour rest period every afternoon and ten hours in bed at night was prescribed A diet only slightly higher than her basal requirements was advised

For nearly three years her status has remained unchanged The heart is still greatly enlarged (Fig 68A) but she has managed to be about on *the same restricted program although the slightest increase in activity* causes marked dyspnea

Over half of the patients who have mitral stenosis develop high blood pressure when they pass the age of 45 which is an interesting fact when we consider that the blood pressures of the younger patients with mitral stenosis tend to be subnormal Does the long standing rheumatic disease produce organic changes that eventually result in hypertension? Certainly the mitral lesion in these patients cannot be ascribed to the hypertension since stenosis of this valve is never produced by an arteriosclerotic process

It is a very remarkable fact that the blood pressure in this patient is continually maintained at levels well over 200 systolic in the presence of an advanced mitral stenotic lesion However there is no reason why the factor that produces hypertension cannot be operative when the patient with the proper hereditary background reaches the age of 45 irrespective of the presence of a mitral stenosis

Levine s observations [18] indicate that the development of hypertension may actually be a helpful mechanism in these patients who have mitral stenosis He points to the fact that the majority of them have a life expectancy below 50 unless hypertension develops The hypertensive process dilates the left ventricle and this tends to counteract the effect of the rheumatic mitral lesion In this way there may be a balance restored between the mitral stenosis and the hypertension since the mitral lesion causes a right sided and the hypertension a left sided hypertrophy Con sequently when failure arises from a prolonged strain on the right side of the heart in mitral stenosis, the beneficial effect of a process tending to produce a left sided hypertrophy can be understood

## Rheumatic Heart Disease with Lesions of All Valves—Prolonged Course of Therapy—Autopsy

**Case 17** Mrs I M a white housewife of 40 had many ho p tal admissions over the course of five years because of recurring attacks of congestive failure Her chief com plaints were shortness of breath and swelling of the abdomen There was no history of rheumatic *fever in childhood*

PHYSICAL EXAMINATION There was marked dyspnea slight cyanosis and ascites The latter increased in amount on each admission Toward the end abdominal taps were performed every second week and the average amount of fluid removed was 5000 cc On the last admission there was also fluid in the right pleural cavity

The heart showed considerable hypertrophy The right base measured 5 0 cm and the left 14 0 cm Systolic thrills and systolic and diastolic murmurs were present over the aortic pulmonic and mitral areas The rhythm was totally irregular with a rate of 100 to 1 0 BP 108/80

CLINICAL DIAGNOSIS A Etiologic Rheumatic Inactive B Anatomic Cardiac enlargement Mitral aortic and pulmonic regurgitation and stenosis C Physiologic Congestive cardiac failure Auricular fibrillation D Functional Classification Class 4 Therapeutic Classification Class E

AUTOPSY Dilatation of the heart especially of the auricles The right ventricle occupied the entire ventral surface of the heart The coronaries were normal The mural endocardium was thick white and opaque in the auricles The mitral valve showed typical fish mouth orifice with greatly thickened valve leaflets The auricular surface was studded with calcific excrescences the size of a pin head and organized vegetations The papillary muscle was fibrosed The aortic cusps were shortened and thickened with dense adhesions at the commissures creating marked stenosis and insufficiency (Fig 69A) Moderate stenosis was present at the pulmonic valve and the cusps presented rolled edges slightly adherent to the commissures (Fig 69B) The tricuspid valve leaflets were thickened and densely adherent with the free edges rolled The chordae tendinae were shortened and thickened

**Discussion** Involvement of all the valves of the heart is rare When an organic tricuspid lesion is present the mitral and aortic valves are usually the seat of advanced changes consequently it is often difficult to evaluate the effects of the tricuspid regurgitation and stenosis on the heart

In this patient separate thrills and murmurs were recognized over the mitral aortic and pulmonic areas but the findings as is quite often the case were confusing over the tricuspid area Systolic and diastolic murmurs were heard over the lower end of the sternum but it was felt that these were transmitted from the mitral area When more than one valve of the heart is damaged by the rheumatic infection the valves on the left side of the heart are as a general rule involved earlier and to a greater degree than those on the right side The order of frequency is usually mitral aortic tricuspid and pulmonic

The course of the disease in this case was typical [106] Hospitalization was required many times during the five years that symptoms were present The patient received active treatment on each occasion and sufficient circulatory balance was restored in a short time to permit her discharge However she did not follow a strict regime between admissions There was always a mild degree of congestion present The easily provoked dyspnea the mild cyanosis the distended jugulars the marked ascites and the chronically engorged and pulsating liver should have suggested a tricuspid lesion The auricular fibrillation present in this case was also consistent with the diagnosis

Between attacks of congestive failure these advanced circulatory alterations caused the patient very little discomfort Her margin was a very slim one and the balance difficult to maintain since the slightest over

exertion very quickly accentuated the cyanosis, dyspnea and other evidences of venous stasis

The management of a patient of this type is described on page 112

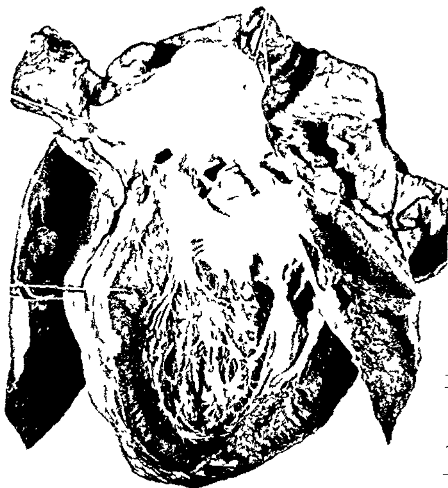


Fig 69 A Rheumatic heart disease Note marked distortion of the aortic cusps (Autopsy No 7 944 Philadelphia General Hospital)

## Rheumatic Heart Disease and Central Nervous System Syphilis—Management

**Case 18** L H an American salesman of 3? was first seen on March 30 1939 complaining of increasing shortness of breath and dull pain over the heart of six months duration For the past two months *he noticed occasional shooting pains in the legs* and nervousness The past history was negative for rheumatism Chancre at the age of ?0

Physical examination showed irregular pupils; the right was larger than the left but both failed to react to light All superficial reflexes were absent even upon reinforcement

Heart Enlarged L B 11 5 cm from the midclavicular line Right base 3 0 cm from M C L Rough systolic and long rumbling diastolic murmurs were heard over the apex A louder than P Thrill at the apex Rhythm regular (Fig 70 A)



Fig 69 B Chronic rheumatic endocarditis of the pulmonary valve Note thick ned r ll l d ges slightly adherent t o mmissur s (A top s No 7 944 Philadelphia General Hospital )

LABORATORY DATA Blood and spinal fluid Wassermann reactions positive Electrocardiogram (Fig 70B) showed widened P waves otherwise normal

CLINICAL DIAGNOSIS Central nervous system syphilis Rheumatic heart disease with cardiac enlargement and mitral stenosis

DISCUSSION Although at times rheumatic may be mistaken for syphilitic aortic disease (Case 30) and in some instances it may be impossible to

determine the exact cause of regurgitation at the aortic orifice (Case 27), this patient should have caused no diagnostic difficulty

In the first place the physical signs elicited on cardiac examination point to involvement of the mitral valve since an apical diastolic murmur accompanied by a sharp snapping first sound are usually characteristic of

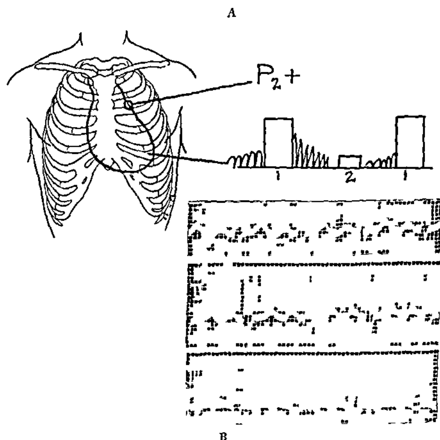



Fig 70 A Chart showing type and location of murmurs
B The electrocardiogram Note increase in the duration of the P waves

mitral stenosis This lesion is always rheumatic Diastolic murmurs at the apex may at times be associated with a syphilitic aortic regurgitation (Austin Flint murmur) In these cases the first sound of the heart will lack the snapping quality of mitral stenosis and the evidence of a marked aortic regurgitation will also be present

Again the age of the patient is a point in favor of the diagnosis of rheumatic heart disease Although it is possible to have clinical signs of cardiovascular syphilis at this patient s age the majority of cases are seen after 35

The presence of a definite history of syphilitic infection and the absence of a rheumatic history are both compatible with the diagnosis of rheumatic heart disease since about 50 per cent of the patients of this age who have established mitral stenosis will not give a history of any form of rheumatic infection

It may be possible that there is a combination of syphilitic and rheumatic heart disease in this patient, for this association is now believed to be a little more common than was once thought However we have nothing whatsoever upon which to base the diagnosis of syphilitic involvement in the absence of both systolic and diastolic murmurs over the aortic area The aortic second sound is not accentuated, and careful fluoroscopic examination shows the aortic diameter is not increased There is likewise no increase in the density of the aorta

At the present time there is no treatment required for the heart From the standpoint of the syphilitic infection the evidence points to the need of immediate treatment designed to prevent the progress of this disease in the central nervous system This is quite different from the plan that should be outlined when the cardiac study suggests an early spirochetal invasion consequently an understanding of the findings and prompt recognition of the type of heart disease present are extremely important

Under proper therapy for neurosyphilis this patient s progress should be good Careful routine cardiac studies should be carried out and future therapy directed toward protecting the cardiovascular system (page 214) This patient offers no cardiac contraindication to the use of pyrexial treatment for his neurosyphilis However in older patients with degenerative cardiovascular disease as the complicating lesion tryparsamide is the drug of choice

# 4

# PERICARDITIS

Pericarditis undoubtedly occurs more often than is recognized It may be acute or chronic involving only a small area of the pericardial sac and escaping clinical detection or it may be a readily recognized process demanding prompt and skillful management

## ACUTE PERICARDITIS

### Etiology

Pericarditis is usually encountered as a complication of some primary infectious process caused by pyogenic cocci (the *Pneumococcus Staphylococcus Streptococcus*) or the organism responsible for the acute rheumatic state While the pericardium may be the site of a primary invasion by the tubercle bacillus this organism more often arrives in the pericardium by extension from a neighboring focus The involvement of the pericardium encountered in uremia cancer and terminal conditions is probably not infectious although the exact cause is unknown in many cases Following a coronary occlusion involving the outer wall of the heart a local inflammatory area may develop in the adjacent pericardium Rarely trauma may be the exciting agent in pericarditis

Acute fibrinous pericarditis is most commonly seen as a complication of acute rheumatic fever and its frequency will depend on the incidence of rheumatic disease in the community A serofibrinous exudate may occur in all forms of pericarditis except those that complicate acute coronary occlusion and uremia In rheumatic infection the fluid that comprises the pericardial effusion is generally clear but may have a slight turbidity while following the invasion of pyogenic organisms a purulent pericardial exudate appears A bloody effusion (hemopericardium) suggests either malignant disease or tuberculosis Air may be present with the effusion (pneumopericardium) when perforation accompanies malignancy of a neighboring organ or it may enter from a pneumothorax or during a paracentesis

Acute pericarditis may be encountered in patients of any age but it is usually seen in young people because of the frequency of acute rheumatic infection at this time of life

### Pathology

When the pericardium is inflamed the membrane rapidly loses its glossy appearance owing to the changes that occur in the epithelial surface Fibrin then appears and the visceral and parietal layers may become adherent

although a solid union is usually prevented by the cardiac action When the layers of the pericardium are separated at autopsy the typical picture known as the bread and butter pericardium is observed (Fig 71) Healing may take place with only a thickening of the pericardium or in cases where the inflammatory process has been widespread adhesions may develop obliterating the pericardial sac or in rare cases anchoring the heart to neighboring structures

## Symptoms

When the inflammation extends from the pericardium to the diaphragm and pleura pain usually results This symptom may first call attention to



Fig 71 Acute fibrinous pericarditis (Hairy heart or cor villosum)

the chest and careful examination at this time will reveal a friction rub While this valuable sign may be absent its presence may remain undetected unless the physician is constantly alert and makes repeated examinations The friction sound is usually grating and harsh and appears to be nearer the ear than the ordinary heart murmur Firm pressure with the stethoscope may increase its intensity It is usually first heard toward the base of the heart and along the left sternal border and is present during

systole and diastole If loud this friction sound may be palpable The presence of a friction rub by no means rules out the possibility of fluid in the pericardial sac, although it usually disappears or becomes diminished in character when an effusion develops

## Signs

Accumulation of fluid in the pericardial sac in amounts in excess of 500 cc usually attracts attention to the heart The earliest sign can usually be detected by a roentgen study since bulgings first appear in the lower part of the cardiac silhouette These changes may also be observed by fluoroscopy As the effusion develops the area of cardiac dulness increases and additional signs become evident chief among which are the absence of cardiac pulsations under the fluoroscope and the characteristic shape of the cardiac silhouette (see Fig 25)

If fluid is present in large quantities it may compress the lung and produce an area of dulness in the left chest posteriorly just below the angle of the scapula Bronchial breathing will be heard over this area (**Ewart s sign**) Unless pericardial effusion is kept in mind these physical signs including the elevated temperature and the dyspnea, suggest a diagnosis of pneumonia

With further increase in the amount of fluid in the pericardial sac the heart sounds become muffled The return venous flow into the right auricle is hindered by the elevated intrapericardial pressure and distention of the jugulars and enlargement of the liver appear There is a fall in the blood pressure and pulse pressure During inspiration the radial pulse may dis appear (**pulsus paradoxus**) *As the venous pressure rises* the heart rate increases to compensate for the decreased diastolic filling The patient becomes cyanotic the veins in the neck are distended and liver engorge ment causes pain in the abdomen to appear there is a fall in the blood pressure and the respiratory rate is rapid The patient leans forward seeking relief This acute distress or **cardiac tamponade** is caused by large collections of fluid in the pericardial sac and calls for emergency treatment

## Paracentesis of the Pericardial Sac

There are several methods of approach to the pericardial sac and it is well to be acquainted with the technic of each one for in the event that adhesions block the approach to a fluid collection in one direction, another avenue may be employed successfully

Pioneers in this field recommended and used the epigastric route and considered it more practical and less dangerous Others use the fifth left *intercostal space about 2 cm inside the left border of percussion dulness* The pericardial sac may also be reached by passing the needle close to the sternum in the fifth left intercostal space to avoid the left internal mam mary artery (Fig 72)

Technic For the epigastric route the patient is placed in a r clining position preferably at an anrle of about 30 degrees with his back v ell supported The skin to the left of the xiphoid is sterilized with iodine and infiltrated with a 1 per cent pro caine solution A ?0 gauge 3½ inch ne dle is used attached directly or by means of a flexible piece of rubber tubing to a 100 cc syrinre The latter is preferable The insertion of the needle is in an upward and inv ard direction for a distance of approximately 2½ inches (Fig 73) depend ng upon the build of tle patient and th thickness of the layer

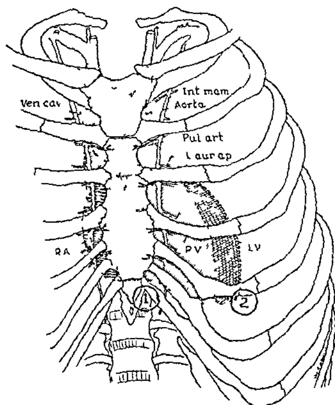
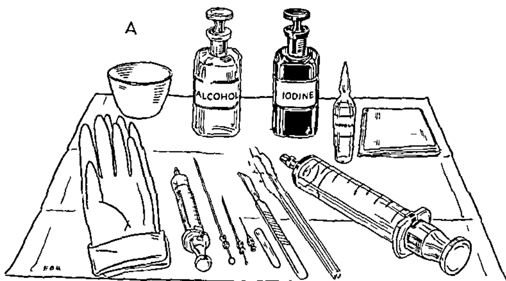
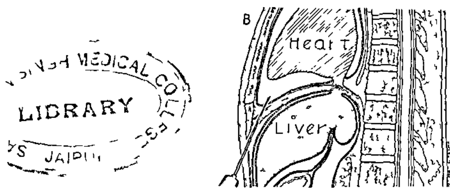



Fig 2 D agram sho inr course of the internal mammary artery and the sites for puncture of the pericar lial sac

of subcutaneous fat The needle enters the pericardium just above the diaphragm from which posit on goo l depend nt drainage may be read ly obtained It necessary the s me site may be used for drainage by res ct on of portions of the fifth and sixth cart lages It is log al to drain an effu ion from its lowest position belo v tle heart; there are no vessels to cause conce n in the area traversed by the exploring needle and the pleural and periton al surfaces are avo ded The adv antages of the piga tric route are all the more evident if purulent pericardial collect ons are su pected

If the anterior approach is used th best point to enter is in the fifth interspace about 1 inch ns de the outer bord r of card ac dulness Procaine solution should be nfiltrated carefully and the nee lle inserted slowly in a backward and upward d rection The needle will be felt to enter the per cardial sac when inserted about 3½ cm but this d st nce w ll arain vary in accordance with the th ckne s of the subcutaneous tissues In th s loca



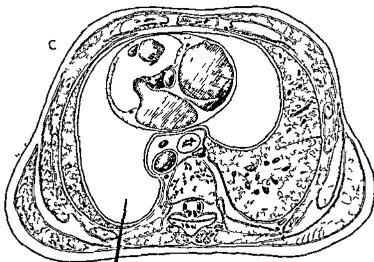




FIG 73 Pericardial paracentesis A Essential equipment B Epigastric approach C Posterior approach (See text for explanation)

tion it is well to use a blunt needle so that no harm may result if a coronary vessel is encountered If the heart is felt against the exploring needle this should cause no concern

Pericardial paracentesis may be carried out with the suction apparatus used for thoracentesis (see Fig 42) At times in the presence of tuberculous involvement when subsequent roentgen studies are to be made small quantities of air are injected into the pericardial sac following the removal of fluid The air outlines the sac sharply and in addition may be valuable in the treatment of the tuberculous process [191] However it is unlikely that the air is of importance in the prevention of subsequent adhesions in the pericardial sac (page 177)

Puncture of the pericardial sac is not a dangerous procedure when carried out with care If the first puncture does not reveal fluid the direction of the exploring needle should be changed If this approach is likewise unsuccessful the attempt should be made to enter the pericardial sac at one of the other sites described above I have often obtained fluid by using the epigastric route when the others have failed [306] In the presence of the dense adhesions that usually accompany tuberculosis of the pericardium the fluid may be encapsulated in which event attempts to reach it in one direction may be unsuccessful whereas large collections may be drained at another site of puncture (see Fig 79)

**In children** in whom an inflammatory process permits greater distention of the pericardial sac the lung may be pushed to one side or displaced and the pericardial surface will come in contact with the chest wall posteriorly If characteristic signs developed posteriorly this region should be selected for puncture (see Fig 73C) Williamson[404, 405] recommends that pericardial paracentesis should be used more often to relieve discomfort and guard the patient against the danger of sudden death He uses the blood pressure changes as a guide Sutton[306] likewise recommends frequent tappings of the pericardial sac by the posterior route and shows that it is possible to carry out the procedure without passage through lung tissue If the posterior route is used the needle should be inserted in the center of the area of bronchial breathing usually about the eighth intercostal space (although the ninth or seventh interspaces may also be used) a little nearer the axilla than the spine Since the fluid obtained is usually thick and coagulates quickly a large bore needle is recommended

### Surgical Drainage

(See page 174)

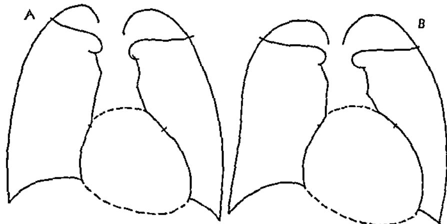
## TUBERCULOUS PERICARDITIS

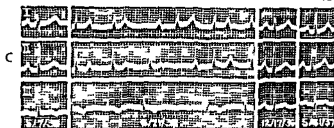
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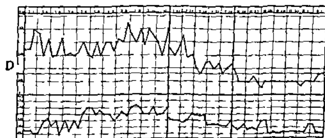
## ILLUSTRATIVE CASES

### Acute Fibrinous Pericarditis During the Course of a Rheumatic Infection

**Case 19** F R a 10 year-old school girl was admitted to the Woman s College Hospital on May 6 1936 complaining of dyspnea and fever



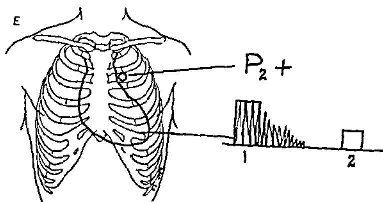




Fig 74 A The orthodiagram at time of discharge (September 1 1936) Note cardiac enlargement chiefly left ventricular B Six months later Note increase in heart size and mitralization
C The electrocardiogram (For explanation see text)
D The temperature and pulse record
E Chart representing clinical findings

HISTORY Acute rheumatic fever two years prior to admission The patient returned to school in two months and was well until the spring of 1936 At this time she visited her family physician who said that she was anemic and underweight and ordered the extraction of several teeth Two weeks following this procedure fever was present every evening A week before admission there was malaise and soreness in the muscles of the legs and the patient was forced to remain in bed When the fever increased and dyspnea developed she was sent to the hospital

PHYSICAL EXAMINATION showed a pale underweight child of 10 suffering from moderate dyspnea T 10 P 130 R 35 B P 110/80 Jugulars not distended The apex impulse was 1 cm outside the midclavicular line No thrills There was a musical systolic murmur over the mitral area The liver was not palpable No edema

LABORATORY DATA Blood count hemoglobin 60 per cent (Sahli) R B C 3 400 000 W B C 16 000 differential P 80 L 0 The electrocardiogram on 5/7/36 (Fig 74C) showed slight elevation of the S T intervals in lead and 3

CLINICAL DIAGNOSIS A Etiologic Rheumatic Inactive B Anatomic Cardiac enlargement Mitral regurgitation Mitral stenosis (?) Acute pericarditis (?) C Physiologic Sinus tachycardia D Functional Classification Class 3 Therapeutic Classification Class E

DISCUSSION At the time of the recurrence of the rheumatic infection this child should not have been allowed to continue at school Again anemia and underweight does not constitute a diagnosis although it is one very frequently made Needless to say the symptomatic treatment of iron and high caloric diet does not lead very far if the underlying rheumatic infection is not suspected and bed rest promptly instituted To add insult to injury but no doubt with the thought in mind of aiding the anemia dental extractions were advised The operation precipitated the attack with a violence that forced the patient to bed and it may have been the time when more extensive cardiac invasion took place

When admitted to the hospital the child appeared acutely ill The degree of fever and the marked dyspnea suggested the diagnosis of pneumonia to the intern in the receiving ward An incorrect diagnosis of this type is made more readily when a pericardial effusion compresses the left lung The appearance of an area of dulness in the left chest below the angle of the scapula over which bronchial breathing can often be heard may confuse the picture further Levine [18] suggests that this syndrome may explain the frequent history of childhood pneumonia in rheumatic patients

However this patient s lungs were clear at the time of admission and when the long history of fever was elicited the cardiac abnormalities helped to establish the diagnosis

The electrocardiogram showed a slight elevation of the S T segment but not enough to suggest the diagnosis of pericarditis * The P R intervals measured 0 2 second While this may be a perfectly normal conduction time in an adult 0 18 second should be viewed as the upper limit of normal in a child of this age

Two days following admission a friction rub was heard over the precordium and was especially loud along the left sternal border This was

* Elevation of the S-T interval should exceed 0 1 mv to be considered abnormal

followed in 24 hours by further prolongation of the P-R intervals (Fig 74C) Frequent dropped beats were detected in the pulse and on auscultation over the precordium no premature beats could be heard during the pauses

The treatment for this patient during the first two days consisted of bed rest in the Fowler position and codeine sulfate 15 mg (¼ grain) by mouth every three hours for pain Since an ice bag over the precordium gave the patient considerable relief it was used continuously

The patient now appeared quite ill The pulse rate mounted with the temperature and the respirations became rapid and shallow In the presence of such an overwhelming cardiac infection digitalis was not given since no symptoms of congestive failure were evident The friction rub disappeared in 36 hours but the heart sounds remained distant although no other evidence of fluid accumulation in the pericardial sac appeared

With the disappearance of chest pain at the end of the first week acetyl salicylic acid 0 6 Gm (10 grains) with an equal amount of sodium bicarbonate were given every three hours in place of the codeine The heart rhythm became regular and the temperature curves approached lower levels (Fig 74D)

A more liberal diet was allowed as the temperature gradually returned to normal at the end of the fourth week At the end of the eighth week the patient was transferred to her home where she remained in bed another month under careful nursing care During this time she gained ten pounds in weight and had no recurrence of fever

When re examined in six months the electrocardiogram (Fig 74C) showed no conduction defect and the rheumatic infection appeared to be inactive However a faint diastolic murmur of aortic regurgitation was heard along the left sternal border although the pulse was not of the Corrigan type An orthodiagram at this time showed a well established mitral stenosis and cardiac enlargement (Fig 74B) The exercise tolerance of the patient was good although continued restriction had given her little opportunity for much exertion

This patient s history reveals the severity of the symptoms that usually accompany acute rheumatic pericarditis Extensive myocardial invasion was shown by the conduction defect that appeared in the electrocardiogram The endocardium by no means escaped since an aortic lesion was discovered at the follow up examination Evidence of the pericardial involvement appeared in the form of the friction rub that was audible for 36 hours All signs of pericardial involvement were absent at subsequent examinations although it is too early to state with certainty that a chronic pericarditis will not develop in later years While many still believe that rheumatism may be responsible for chronic constrictive pericarditis White[398] refers to a group of 100 children with chronic rheumatic heart disease followed over a 10 year period at the House of the Good Samaritan in Boston among whom not a single case of constrictive pericarditis has developed

### Acute Suppurative Pericarditis Secondary to Type II Pneumococcus Pneumonia—Drainage—Recovery

Case 20 J V [illegible] girl [illegible] 13 was seen on 1 4 38 complaining of cough and fever

History Ten days prior to admission to the hospital the patient developed fever malaise and cough Four days later she had a chill followed by pain in the right lower chest and upper abdomen The pain was increased by breathing

Physical Examination T 101 P 110 Dyspnea marked Chest expansion was diminished on the right There was dulness to flatness in the right chest posteriorly below the angle of the scapula The breath sounds were diminished and there were crackling rales heard over the same area The vocal fremitus was markedly increased and the breath sounds in the left chest were exaggerated

The cardiac apex was in the fifth interspace in the midclavicular line The rhythm was regular No murmurs were heard There was tenderness over the right upper abdominal quadrant

Laboratory Data Blood count hemoglobin 80 per cent (Sahli) R B C 4 400 000 W B C 1 000 P 88 L 10 M Sputum pneumococcus type II

Roentgenogram (1/9/38) (Fig 75) showed a uniform density extending from the diaphragm to the fifth rib in the scapular line on the right side The heart and aorta were displaced slightly to the left The left lung was clear The changes present in the right lung indicated consolidation involving the lower and middle lobes In addition there was a moderate amount of free fluid in the right pleural cavity

Course These findings on admission indicated the presence of a type II lobar pneumonia Wide excursions of the temperature prompted us to aspirate the right chest on the seventh day Pus was obtained Consequently an incision was made under local anesthesia in the posterior axillary line paralleling the ninth rib The periosteum was stripped 1 inch of the rib removed the pleural cavity opened and a large quantity of pus evacuated A drainage tube was inserted

A week later the pulse rate began to rise and there was noted an increase in the cardiac dulness both to the left and to the right The heart sounds were distinct the first sound at the apex was split and a soft systolic murmur was heard in this region An area of percussion dulness appeared at the left base (Ewart s sign) a slight distention of the jugular veins was noted and this was followed by an increase in the respiratory rate A diagnosis of acute pneumococcic pericarditis was made

A roentgen ray examination at this time 1/18/38 (Fig 75) showed a pneumothorax on the right and a considerable increase in the size of the cardiac silhouette There were signs of fluid in the pericardial sac

Paracentesis of the pericardium (page 164) revealed 300 cc of a turbid fluid and a laboratory examination of the stained smear showed gram positive diplococci in pairs

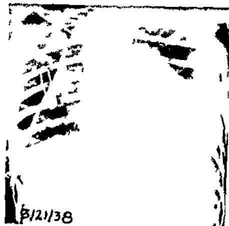
Discussion (Dr James Lehman) *—In this patient the pneumococcus invaded the pericardium from the empyema cavity Infectious processes below the diaphragm such as peritonitis abscess of the liver or pancreas ulcer of the stomach and subdiaphragmatic abscesses rarely extend to the pericardium

The symptoms that follow the involvement of the pericardium are variable and frequently are obscured by those of the underlying condition Consequently unless the complication is suspected and the symptoms searched for suppurative pericarditis may run its entire course without being recognized

The distinctive to and fro pericardial friction sound that has been

* Associate in Clinical Surgery Woman s Medical College of Pennsylvania













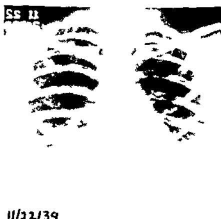



Fig 75 A Series of roentgen films taken during course of illness 1/9/38 Pneumonia with large pleural effusion 1/18/38 Pneumothorax following aspiration of large amount of fluid Collapse of the right lung 2/21/38 Enlargement of the cardiac silhouette Note the re expansion of the right lung 3/21/38 Drainage tube in pericardial sac 4/20/38 Cardiac shadow decreasing Patient discharged 11/22/39 Over a year later Heart smaller Physical condition of the patient excellent

described should always be sought at every examination I believe it may be heard during the early stages in nearly all cases When present it is missed most often because the integrity of the pericardial sac is not suspected so it should be a clinical rule to make frequent examinations of the precordial area in every case of pneumonia particularly if empyema has already developed

You will note from the history that this patient had some reference of pain to the abdomen At one stage abdominal tenderness was present Not infrequently pain referred to abdominal areas from a lower lobe pneumonia simulates an acute abdominal condition Sometimes in children a

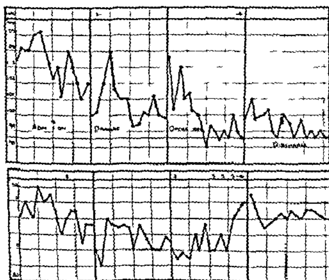


FIG 75 B Temperature and pulse record

pericardial involvement will produce pain in the abdomen that may be diffuse or it may be localized in the right lower quadrant and simulate acute appendicitis In many of these children most often rheumatic in type pain will also be referred to the neck or to the shoulder The development of the signs of pericardial involvement often clears the picture but sometimes the differentiation is not easy

When pericardial effusion increased in this patient typical signs developed Paracentesis of the pericardium revealed the presence of pus and indicated immediate surgical interference The operation of preference in these patients is resection of the fourth fifth and sixth ribs on the left side (Figs 76A and B) Adequate exposure is necessary Care should be taken first to avoid injury and contamination of the pleura and second to provide dependent drainage

The drain should be placed inside the pericardial sac so that all areas

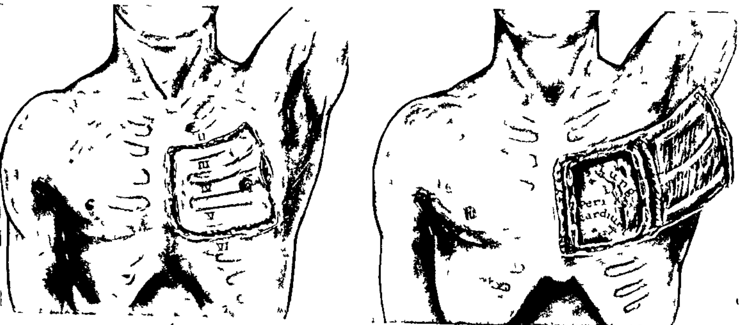



Fig 76 Surgical drainage of the pericardial sac A Incision B Flap turned back and pericardium exposed

will be drained particularly the posterior section If a walled-off abscess is overlooked a fatality may result Gentle irrigation is not dangerous if small quantities of warm sterile physiologic saline are used in fact these may be quite necessary when the pus is thick and plugs of fibrin clog the drainage tube

If aspiration alone is used and operation delayed the mortality in acute suppurative pericarditis is 100 per cent If adequate drainage is established half of these patients survive Only 5 per cent of the cases that recover subsequently develop fatal adhesive pericarditis [339]

Following the adequate drainage of this child's pericardium and the subsequent use of sulfapyridine (see page 198) the temperature gradually returned to normal and the remainder of her convalescence was uneventful

A follow up examination nine months after discharge showed very slight thickening of the pleura at the base of the right chest with obliteration of the right costophrenic sulcus by adhesions (Fig 76) This portion of the chest therefore is almost normal and gives very little evidence of the extensive involvement that was present The heart is still displaced and slightly enlarged but its contour is normal The patient has no complaints and her exercise tolerance is good

## Tuberculosis Pericarditis—Autopsy

**Case 21** E A, a colored male of 31 was admitted to the Philadelphia General Hospital on 4/ 9/ complaining of cough shortness of breath and swelling of the legs

History The patient had attacks of pneumonia at the ages of ?0 7 and 30 Following the last attack he began to notice dyspnea and cough There was considerable weight loss Four months before admission swelling of the legs appeared and this was followed by a considerable increase in the dyspnea A month before admission the abdomen began to swell

Physical examination showed B P 116/80 dyspnea cyanosis and anasarca The heart rate was 90 the rhythm regular The heart sounds were weak and distant No murmurs were heard There were signs of fluid at both pulmonary bases A few crackling rales were heard over the left apex anteriorly Marked ascites was present

Laboratory Data Wassermann negative Blood count Hemoglobin 68 per cent (Sahli) R B C 3 ?00 000 W B C 7 00 differential normal Urine specific gravity 1 028 light cloud of albumin with a few hyaline casts

Electrocardiogram inverted T1 and T

Chest roentgenogram Bilateral pleural fluid

Course During the patient's stay in the hospital chest tap was performed 15 times and a total of 500 cc of pale amber fluid removed He gradually lost ground and died three months after admission

Clinical Diagnosis Pick's disease

Autopsy (Fig 77) Heart weight 1010 Gm The epicardium was markedly adherent to the pericardium There was a zone between the pericardium and the epicardium varying in width which contained communicating pockets that were filled with a cheesy material Here and there was a suggestion of the presence of the same material in the myocardium The muscle of the right auricular appendage showed definite tuberculous myocarditis The left lung showed a moderately advanced fibro ulcerative tuberculosis

**Discussion** Tuberculous pericarditis is not a rare condition Since it occurs in about 5 per cent of autopsies on tuberculous subjects it may be

considered a disease of decided clinical importance While the mortality is high, healing may take place in some cases with the subsequent development of chronic constrictive pericarditis



FIG. 77 Tuberculous pericarditis and myocarditis (Autopsy No 7 50 Philadelphia General Hospital )

The pericardium may be invaded from foci in the lungs peritoneum pleura or *mediastinal nodes* The *most common* primary focus is the lung The patient whose history appears above i mple of this type Very

rarely the pericardium is the only seat of involvement brought to light at autopsy. Many cases thought to be examples of primary tuberculous pericarditis however may have an original focus so small that it is easily overlooked. The infection of the pericardium in some cases may be part of a general miliary tuberculosis in which event it reaches the pericardium by way of the blood stream. In the presence of a miliary tuberculosis the pericardial involvement is nearly always impossible to demonstrate clinically.

Direct extension from the lungs is probably not as common an occurrence as invasion of the pericardium by way of the lymph channels that drain the mediastinal glands. Symptoms arise during the course of this




Fig 78 Tuberculous pericarditis

A Before paracentesis. The cardiac silhouette is greatly enlarged due to collection of fluid in pericardial sac.

B Following paracentesis of 900 cc and air injection. Note small heart and thickened pericardial sac. (Courtesy X Ray Department Philadelphia General Hospital.)

involvement and lead to its detection for it may be ushered in by a friction rub. In this respect it is not unlike the rheumatic variety. The symptoms of the pulmonary disease may in some cases obscure this early sign; in others no symptoms of any kind are present. Effusion may attract attention particularly if it is large, in which event dyspnea enters the picture. The apex beat will no longer be palpable and the heart sounds will become distant and muffled and percussion of the chest will reveal a widened area of cardiac dulness. Pulsus paradoxus appears if the effusion is large and bulging of the neck veins will be evident on inspection. A paracentesis of the pericardium at this stage with the discovery of a hemorrhagic type of fluid arouses considerable suspicion concerning the nature of the process. Success in the demonstration of tubercle bacilli in the effusion, either directly in the stained smear or following guinea pig inoculation, will clinch the diagnosis.

If air is injected into the pericardial sac following paracentesis (Fig 78) a small heart will usually be revealed surrounded by a very much thick

ened pericardium This appearance is characteristic of the lesion and is second in importance only to the finding of the tubercle bacillus

*Since no invasion of the endocardium occurs in tuberculous pericarditis* no murmurs are usually heard This may aid many times in differentiating tuberculous from rheumatic pericarditis since in the rheumatic state characteristic murmurs are nearly always present In acute rheumatism *there is a leukocytosis while in tuberculosis there is usually a normal white* blood cell count or even a leukopenia A higher temperature curve and a tendency to the appearance of congestive failure suggest rheumatic disease

Although in the early stages it may be difficult to differentiate between

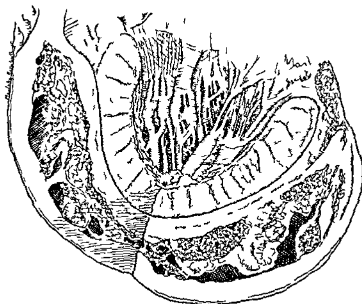


FIG 79 Tuberculous pericarditis Note the presence of exudate and adhesions between the thickened layers of pericardium

the acute rheumatic and the tuberculous forms of pericarditis subsequent happenings may make the distinction possible The rheumatic fibrinous exudate may disappear or pass into a chronic type with adhesions In *tuberculosis the pericardium becomes thickened the effusion becomes* less and masses of organized exudate remain between the layers of pericardium (Fig 79) Eventually adhesions may contract and this whole area may become obliterated with the development of a chronic fibrous process In the ashes of the fire no structure sufficiently characteristic to identify the original process as tuberculous may remain The absence of a rheumatic history or lesion and the presence of a tuberculous focus at another site in the body may be the only evidence that suggests the real nature of the initial process

In Fig 77 the tuberculous infiltration of the myocardium served to

establish the identity of the pericardial lesion The appearance of calcium in the healed pericardium does not establish the process as tuberculous since this type of infiltration often occurs in the healing of suppurative conditions

In tuberculosis of the pericardium larger amounts of fluid are recovered by paracentesis than in any other type The injection of air into the pericardial sac at the time of the tap was first suggested by Wenckebach in 1910 He replaced the fluid removed with half the volume of air and his patient showed much slower reaccumulation of the effusion Subsequent reports seemed to confirm this view and the first series of cases reported in the literature showed great symptomatic relief It is easier for the heart to work against air than a solid blanket of fluid and this decrease in *resistance may be responsible for the improved state of the circulation* Air however will not prevent the formation of adhesions in the sac as claimed by the original investigators in this field

The general measures used in the treatment of tuberculosis in other locations are also employed in tuberculous pericarditis Relief of intrapericardial pressure by tapping as often as necessary absolute bed rest and a high caloric diet constitute the essential points in the program Digitalis is rarely indicated since the symptoms of cardiac embarrassment are caused by the tamponade

If healing is accomplished and the signs of chronic constrictive pericarditis develop the only satisfactory treatment is surgical (page 184) The best results are obtained in young people who have no evidence of active infection

## CHRONIC PERICARDITIS

The resolution that follows acute pericarditis pursues a variable course depending on the type of the initial involvement and to a great extent upon the severity and duration of the process The clinical detection of chronic pericarditis is difficult for many times it produces no signs or symptoms and in the majority of the cases its presence does not influence the future health of the patient

### Signs and Symptoms

As healing of the pericardial lesion takes place it quite often erases all evidence of the nature of the primary process Consequently unless the past history of the patient is typical much confusion is bound to exist at the bedside or at postmortem in regard to etiology However evidence of rheumatic infection or tuberculosis elsewhere in the body may be of assistance while more rarely pneumonia coronary disease and malignancy must be considered as possible causes In the northern states rheumatic infection is found most often in the past histories of the patients presenting symptoms of chronic pericardial disease

The isolated patches of fibrous tissue in the pericardium pointed out as 'soldiers spots' by the pathologist deserve only the passing interest that is usually given to them. Even if definite adhesions exist between the layers of the pericardial sac no disturbance of cardiac function usually appears. In fact in cases where adhesions are so numerous as to entirely obliterate the space between the two layers of pericardium, the heart may be unaffected if adhesions to neighboring structures are absent.

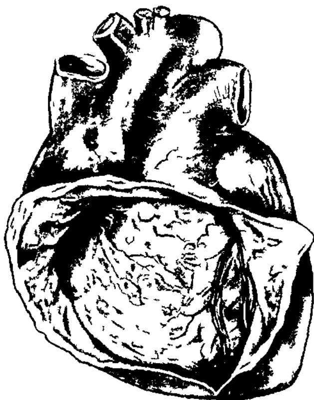


FIG 80 Chronic calcific pericarditis

**Constricting Adhesions and Chronic Tamponade** However if the healing process produces a thick layer of adhesions that entirely surround the heart cardiac action may in time be seriously compromised. Contraction of these adhesions the unyielding character of which meanwhile may be considerably augmented by a widespread calcium (rarely bone) deposition not only interferes with the diastolic filling of the heart but also blocks venous return by constricting the lumen of the entering veins. This condition is known as chronic constrictive pericarditis and is a feature of Pick's disease (Fig 80).

Adhesions may likewise develop between the pericardium and its con

tents and surrounding structures in the chest wall or mediastinum (chronic mediastinopericarditis) causing angulation or rotation of the heart and in consequence considerable serious interference with its action. Recent views are not entirely in agreement with the old theory that cardiac traction is the cause of the grave sequelae when adhesions develop between the heart and neighboring structures. Symptoms of importance arise only when adhesions either constrict the heart or cause its angulation or rotation by the formation of firm bands between the heart and neighboring structures.

When adhesions constrict the heart and interfere with the venous return the liver enlarges. Direct involvement of the hepatic vein may contribute to the severity of the process and ascites appears (Pick's mediastino pericarditic-pseudocirrhosis). The symptoms of chronic cardiac tamponade develop (Fig 81). It is much easier to remember these signs if the picture of compression of the heart is recalled instead of the old idea that the heart wears itself out to the point of failure by the added work of pulling against the adhesive bands. **The triad of Beck** (1) a small quiet heart (2) a high venous pressure in the arm and (3) ascites and large liver[21] presents a brief summary of this condition and should facilitate the recognition of the disease.

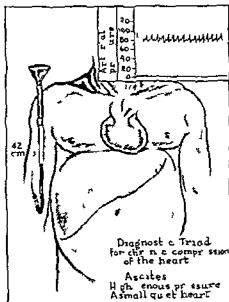



Fig 81 Chronic cardiac compression (Redrawn from Beck.)*

* Reprinted by permission American Heart Journal

It can now be seen that the signs elicited on examination of the patient who has chronic pericarditis depend on the nature and distribution of the adhesive bands. Unless previous rheumatic disease was present the heart will be normal and no murmurs will be heard. If rheumatic involvement is present the characteristic murmurs and cardiac enlargement may be detected. Usually the compressed heart of chronic pericarditis is small and under the fluoroscope its pulsations are not in evidence. If adhesions have securely anchored the heart no change in position will be seen to follow the respiratory excursions. The same fixation may be demonstrated on physical examination. In some instances where the heart is firmly anchored to the chest wall by adhesions a systolic retraction of this area usually in the eleventh posterior interspace may be seen (Broadbent's sign).

## TREATMENT

The treatment of chronic pericarditis rests upon a careful estimation of the amount of embarrassment of the circulation that is produced by the adhesions [8, 30] The effect of previous rheumatic disease on the symptoms of congestion must likewise be evaluated If tuberculosis subacute bacterial endocarditis or other infections are present the degree of their activity should be determined for operation in the presence of any active infection invariably leads to a poor result If the heart was normal before the adhesions developed operation may restore complete cardiac function for an indefinite period The most favorable cases for operation are young individuals presenting the triad of Beck who show no other evidence of heart or vascular disease

## CHRONIC CARDIAC COMPRESSION

(See page 180)

### ILLUSTRATIVE CASES

#### Chronic Constrictive Pericarditis [13]

**Case 22** R F a white school boy of 12 was admitted to the Woman's College Hospital on 5/5/36 complaining of swelling of the abdomen

History Three years before admission the parents first noticed this swelling which gradually became worse until weekly tappings were required Dyspnea and cyanosis on exertion were noted Slight edema of the feet was present three weeks before admission No history of rheumatic infection A maternal grandfather had tuberculosis

Physical Examination Slightly emaciated boy of 1 years B P 90/60 Dyspnea Swelling of the veins of the neck No cardiac enlargement Fixed apex beat No systolic retraction Apical systolic murmur Lungs showed evidence of fluid at the right base Marked ascites The liver was felt 9 cm below the costal margin after tapping

Laboratory Data Roentgenogram showed heart only slightly enlarged Lateral view showed a shell of calcium encasing the heart (Fig 8 C)

The electrocardiogram showed on first examination (See Fig 254A) notched P waves diphasic T and inverted T3 Examination two years later showed decreased voltage of the QRS groups and flat T waves

Clinical Diagnosis A Etiologic Unknown B Anatomic Constrictive pericarditis C Physiologic Restricted diastolic filling N S R D Functional Classification Class 4 Therapeutic Classification Class F

Discussion *This patient when first examined showed ascites* and signs of venous obstruction out of proportion to the amount of cardiac damage that could be demonstrated on physical examination A rheumatic history and signs of organic valvular disease were conspicuously absent The heart was small and the lateral roentgen study (Fig 82) showed a shell of calcium surrounding the heart A comparison of the electrocardiograms (Fig 254) revealed a decreasing amplitude of the QRS complexes and T waves that is so characteristic in these cases

Further studies showed a diminished cardiac output per beat and per minute and *a decrease in the velocity* of the blood flow The small heart




FIG 8 A Swollen jugular veins
B As it s
C Roentgen film (lateral view) Note layer of calcium encasing the h a t

the decreased amplitude of the contraction evident on fluoroscopy and the shell of calcium in the roentgen film suggested that the cardiac symptoms in this child were due to decreased diastolic filling

In view of this explanation for the symptoms it was obvious that digitalis could contribute nothing to the plan of therapy Furthermore symptomatic medical treatment carelessly carried out in these cases may actually be harmful For example venesection performed for the cyanosis and bulging jugulars evident on slight exertion would dangerously lower the venous pressure to the point where it might be insufficient to accomplish auricular filling

The treatment indicated is pericardectomy The result will depend upon intelligent pre operative treatment and the experience of the surgeon The pre operative regime consists of limiting the fluid intake to 1200 cc daily and the sodium chloride content of the diet to 2 0 Gm daily The protein intake should be increased owing to the low serum protein that is generally encountered Mercurial diuretics are useful in the dehydration program that precedes operation Mercupurin is usually given in 2 cc amounts intravenously every three days with ammonium chloride in 1 0 Gm (15 grains) doses by mouth after meals Theobromine sodium acetate in 0 3 Gm (5 grains) doses after meals may be helpful between injections of the mercupurin

Thoracentesis (page 94) and abdominal tapping (page 96) were both indicated prior to operation in this patient Following the pericardectomy the diuretic drugs are continued until a complete cardiac balance is restored An intravenous injection of 2 cc of mercupurin every week for some months can be used to advantage and is not harmful

The operation of pericardectomy is usually carried out under ether anesthesia The second third fourth and fifth costal cartilages are resected on the left side The periosteum is left in place and this permits regeneration of the bony framework of the ribs following operation As much of the pericardium as possible is excised over both ventricles and the heart released from its constricting shell Relief is prompt striking and generally permanent

# 5

# ACUTE AND SUBACUTE BACTERIAL ENDOCARDITIS

Because all the sick do not recover does not prove that there is no art of medicine —CICERO *De Natura Deorum* Bk ii Ch 4 12

Bacterial endocarditis remains one of the saddest chapters in clinical medicine Whether it appears on the scene as a complication of an acute infectious process or whether it pursues a more insidious course after implantation upon a previously damaged heart valve the end result is the same In the first instance the disease is a rapidly fatal one in the second the duration of life may be a year rarely longer allowing the physician ample time to display his therapeutic skill Although the progress in treatment as far as final results are concerned has been slight with the continued improvement in our methods of attack the physician of tomorrow may witness the victory

## ACUTE BACTERIAL ENDOCARDITIS

Acute bacterial endocarditis is still referred to as malignant or ulcerative endocarditis The term ulcerative refers only to the pathologic change seen in the valves and gives no idea as to the nature of the etiologic agent Consequently the disease should be referred to as acute bacterial (pneumococcic streptococcic etc ) endocarditis using in the diagnosis whenever possible the name of the organism responsible for the lesion Acute endocardial lesions of a rheumatic nature are not included in this group

### ETIOLOGY

The organisms most commonly associated with acute bacterial endocarditis are *Streptococcus hemolyticus* (55 per cent) *Pneumococcus* (13 per cent) *Staphylococcus aureus* (12 per cent) *Gonococcus* (11 per cent) while miscellaneous invaders like the *Meningococcus* and the colon anthrax plague and pyocyaneus bacilli make up the remaining small percentage (Thayer) When these organisms are the active agents in the heart infection the clinical course covers but a few weeks If the duration of life is two months or over the condition is more apt to be due to *Streptococcus viridans* and is referred to as subacute bacterial endocarditis

### PATHOLOGY

Most of the bacteria that cause this fulminating type of infection are cocci They attack the heart by way of the blood stream to which they

gain entrance from a number of locations Common avenues of invasion include the uterine wall during the early puerperium the pneumonic lung wounds gonorrheal joints the meninges osteomyelitis boils and the infected pockets that remain following dental extractions Generally the organism gains easier foothold in previously damaged hearts lodging in most cases on the valvular structures although the endocardium is by no means immune to attack The vegetations that are products of bacterial growth may appear on the inner wall of the auricle or ventricle and along the intima of the aorta or a patent ductus arteriosus Their rapid growth gives rise to the appearance of ulceration and the heart valves or aorta may be so extensively invaded and weakened that aneurysmal pouchings (mycotic aneurysms) may soon appear Consequently rupture of these valve leaflets is not unusual

## Signs and Symptoms

**Vegetations** The persistence of the high fever of the original infection often tends to conceal the attack on the cardiac structures Blood cultures taken during the acute illness may have already detected and identified the invader in which event the question of cardiac involvement is often a matter of debate However if the patient lives and the endocardial vegetations increase in size two happenings call the clinician's attention to the heart First the growth of the vegetations on the aortic or mitral valve may change the character or pitch of a murmur previously noted If murmurs were absent one or more may now appear *If these changes in the physical signs appear suddenly ulcerative processes should be suspected* However the character or intensity of the murmur should never be regarded as an index of the severity of the process



FIG 83 Subacute bacterial endocarditis Petechiae on abdomen

**Embolism** The second happening is a more serious one Large (and usually infected) particles of the vegetations may break off form emboli and lodge in some distant organ (kidney spleen brain) with the production of metastatic abscesses Petechial hemorrhages into the skin may appear at this stage and may be regarded as manifestations of the same embolic process (Fig 83) In some cases none of these signs may appear in which event the cardiac complications are revealed only at autopsy

**Laboratory findings** aside from the positive blood culture give little

aid in detecting cardiac infection The leukocyte count is high and the urine may show a moderate amount of albumin and a few red blood cells to suggest a focal embolic lesion The electrocardiogram is negative unless the conduction system is directly invaded by extension from the endocardium which rarely if ever occurs

### Prognosis

In acute bacterial endocarditis embolism is usually the cause of death in a few days or weeks In other cases an overwhelming toxemia may hasten death before the inroads of the cardiac infection have been extensive Less often congestive failure appears as the terminal episode Fatal hemorrhages into the skin and mucous membranes or from the nose or gastro-intestinal tract may take place in some cases Recovery from this disease is most unusual but if it occurs chronic valvular disease will be the sequel

## ILLUSTRATIVE CASE

### Acute Bacterial (Gonococcal) Endocarditis Complicating Puerperium —Autopsy

**Case 23** W B a colored female of 18 was admitted to the Philadelphia General Hospital on 4/8/36 complaining of fever and pain in the left elbow of a week s duration She was pregnant at term

Physical Examination B P 150/100 T 101 F P 1 0 The rhythm was regular The apex beat was in the fifth interspace in the m dclavicular line There was a soft systolic murmur in the region of the cardiac apex

Laboratory Data Wassermann negative The ur ne showed a cloud of albumin and casts Blood count hemoglobin 71 per cent (Sahli) R B C 4 300 000 W B C 19 000 P 76 L 4

Course On 4/14/36 the patient was delivered spontaneously On 4/19/36 the blood cultu e was pos tive for gonococcus The septic temperature continued Death occurred on 4/24/36

Autopsy The heart weighed 350 Gm The septal leaflet of the aortic valve was ulcerated and necrotic and replaced by a soft m ss of grayish red f iable vegetations (Fig 84) The remaining leaflets were normal Along the line of closure of the mitral valve and on the au icular surface there was a single row of fresh vegetations which were easily broken off There was an ulcerat on on the m tral leaflet the size of a match head but no evidence of rheumatic valvul tis was present The tricuspid and pulmonic valves were normal

**Discussion** Considering the prevalence of gonorrhea gonococcal endocarditis is a rare complication In a recent publication Freund and his co workers were able to collect only 139 cases from the literature[109] Males predominated in their series and the age of the patients ranged from two to 51 years The average duration of the disease they found to be about ten weeks and the mortality 93 5 per cent

The diagnosis in this patient was established by blood culture and confirmed when the gonococcus was again recovered from the valve lesions at necropsy

There is at present no general agreement in regard to the plan of treatment that should be adopted in these cases In the few instances of recov

ery from the disease that are on record repeated transfusions, vaccines made up of killed gonococci antigonococcus serum and the intravenous administration of dyes constitute the measures employed However, we can



FIG 84 Acute bacterial (gonococcal) endocarditis of the aortic valve (Autopsy No 31 364 Philadelphia General Hospital)

safely say that all these therapeutic weapons have been used in many patients who did not recover

In the treatment of gonococcal arthritis and other systemic manifestations of the disease vaccines have enjoyed considerable reputation and acute reactions with temperature are essential to success Experimentally it has also been shown that nearly all the gonococci in cultures are killed

when exposed to a temperature of 106° F for four or five hours These facts form the basis of treatment by hyperthermia

Favorable reports have appeared recently in the literature where this type of therapy has been successful in gonococcal endocarditis and septicemia Williams[401, 40] using the Kettering hypertherm reports one case where fever treatment resulted in sterilization of the blood Healing lesions were found at autopsy and death was due to uremia and coexisting syphilis of the liver In another case reported as gonococcal endocarditis with gonococcal arthritis, fever treatment resulted in prompt recovery Freund and his co-workers report a cure in a negress of 20 following the use of fever therapy and recommend 106 7 to 107° F as the optimum temperature for gonococcal endocarditis However Hoyt and Warren[164] report a case treated for 17½ hours at 106° F where the result was fatal Krusen and Elkins[196] have also reported a case of gonococcemia with endocarditis treated by fever therapy without favorable effect

The possibility of a radical cure of the invasion by extirpation of the *focus through which entry is gained into the blood stream is suggested* by Wheeler and Connell who recommend hysterectomy

Today attention centers chiefly on sulfanilamide and its derivatives in the treatment of this disease Much has been written concerning the usefulness of these new drugs in gonococcal infections but opinion has not crystallized in regard to many essential points in therapy Long and Bliss[36] during the past few years have used sulfanilamide in two patients suffering from gonococcal endocarditis and bacteremia In both instances intensive therapy using doses suggested in Table V brought the endocarditis and bacteremia under control Both patients developed signs of acute nephritis during the course of their infection and in one death occurred from this complication One cure is reported following the use of sulfanilamide Long and Bliss point out the necessity of prolonged therapy if recurrences are to be avoided

At the present time the number of cases has been too small to permit a definite opinion as to the exact status of sulfanilamide therapy in acute bacterial endocarditis Other measures including frequent transfusions vaccines dyes and sera have been so uniformly unsuccessful that the hope of the future hinges largely on the success of the sulfanilamide group used either alone or in combination with pyrexial therapy

## SUBACUTE BACTERIAL ENDOCARDITIS

### Etiology

Subacute bacterial endocarditis is caused by organisms that show a less fulminating course after a foothold has been gained on a damaged valve leaflet but reveal an ultimate mortality that is comparable to the acute form Nonhemolytic *Streptococcus viridans* is the invader in the majority (90 per cent) of the cases The influenza bacillus the *Enterococcus* the *Meningococcus* and organisms of the *Brucella* group make up the remaining

percentage Very rarely higher bacteria (*Leptothrix* and *Actinomyces bovis*) invade the endocardial structures and produce a similar clinical picture

Subacute bacterial endocarditis occurs in about 1 to 2 per cent of all cardiac cases and in one out of every 25 to 50 cases of rheumatic heart disease *Any vascularized scar in the endocardium appears to invite implan*tation of the *Streptococcus viridans* Consequently all congenital defects as well as arteriosclerotic and syphilitic valvular lesions, form potential sites Studies on a large series of cases[96 68 383] have shown that the *Streptococcus viridans* usually gains access to the blood stream from foci of infection in the upper respiratory tract and mouth Chronic otitic infections genitourinary tract foci and open wounds should also be regarded as possible portals of entry The gastro-intestinal tract plays a minor role in spite of the many reports emphasizing the possibility of stasis with subsequent invasion from this area

It is not unusual to see cases of quiescent and well healed rheumatic endocarditis start their downhill course following an ill advised tonsillectomy or dental extraction Following these procedures the large areas in the mouth and throat covered by layers of necrotic tissue give ample opportunity to the ever present *Streptococcus viridans* to gain an easy entrance

## Pathology

When infection occurs the mitral valve is involved in the majority of instances but the aortic valve particularly if it is of the bicuspid type is *not infrequently attacked (see Fig 126) The vegetations may also spread* along the walls of the heart chamber invade the aorta and block the coronaries The *S viridans* also shows a great affinity for congenital defects of all kinds

When implantation occurs on a valve the inflammatory growth is again capable of extension into the valve with subsequent weakening of its structure and the formation of valvular aneurysms Vegetations break off and form emboli with the production of further complications in distant regions

## Signs and Symptoms

The symptoms of subacute bacterial endocarditis are much less severe than those of the acute variety that have just been described The onset *of subacute bacterial endocarditis is usually slow and so insidious that the* patient when seen is not able to give the exact date of the origin of his complaints At first a low grade fever with weakness loss of weight anorexia and joint and back pain is present Members of the patient's family or his business associates may notice the pallor and weight loss and suggest medical attention Tuberculosis malaria and other infections are apt to be suspected until a detailed study including a blood culture reveals the diagnosis *Often an embolic episode may first cause the patient*

to seek medical advice particularly if the embolus lodges in the region of the retinal artery or in an abdominal organ By the time that attention is directed to the source of trouble a palpable spleen clubbing of the fingers and marked anemia and all the signs that are so characteristic of the disease are usually present

**Murmurs** If no murmurs are audible care should be used in making the diagnosis If a murmur is already present following an old attack of rheumatic fever its features should be recorded and closely watched If the murmur changes in pitch or intensity during an interval between visits this may be considered as valuable evidence Crops of petechiae should be searched for each day (Fig 84) Tender fingers and toes as well as clubbing are other valuable aids in establishing the diagnosis

**Emboli** Repeated insults of an embolic nature may so decrease kidney function in patients with subacute bacterial endocarditis that uremic symptoms soon appear (Fig 85) Albuminuria and hematuria will appear before this terminal stage is reached and furnish a clue to the presence of renal disease

**Heart failure** occurs more frequently in subacute bacterial endocarditis than is commonly believed a fact that has been brought out in a study by Buchbinder and Saphir[4] These observers likewise have shown that the heart in this disease is the seat of widespread anatomic changes consisting of minute emboli infarcts and abscesses and diffuse areas of inflammation with perivascular fibrosis These widespread changes are important to keep in mind when we formulate any plan of therapy

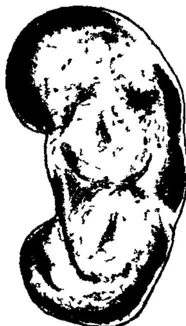


Fig 85 The kidney in subacute bacterial endocarditis Note the numerous petechiae and the scarring caused by multiple infarcts (Flea bitten kidney)

## Prognosis

If the opinions of all physicians who have treated subacute bacterial endocarditis were gathered a uniform hopelessness as to the outcome would be seen to prevail Once the diagnosis is firmly established I have never seen recovery take place Libman[30, 31] reports at least 3 per cent of recoveries in the usual type of the disease and believes that there is a possibility that more recoveries in mild cases occur but are overlooked These mild cases however are difficult to recognize Bacteria in the blood

stream may be few in number or absent altogether owing to their prompt destruction by the body forces of resistance as soon as they are swept from the vegetations If we view the matter from the angle of the postmortem examination evidence seems to be in favor of healing since the pathologist routinely encounters fibrosis and calcification in the structures that are involved in this disease

### Management

The management of these hopeless cases of subacute bacterial endocarditis is generally viewed by the practitioner as an ordeal that must be faced No other situation in medical practice surpasses it in testing the caliber of the physician These patients and their worried families require time thought tact and resourcefulness many times insisting on a trial of various remedies both old and new Continued failure often undermines confidence, and the physician becomes wearied and discouraged when he should remain watchful and hopeful At least we can keep hoping meanwhile guarding our patients against unnecessary and at times actually harmful measures A regime of therapy should be chosen that will encourage and aid the forces of Nature My therapeutic attempts and their uniformly poor results are reflected in the following case histories

## ILLUSTRATIVE CASES

(Showing Hyperpyrexial Therapy Transfusion Sulfanilamide, Sulfapyridine Heparinsulfapyridine Therapy and Arteriovenous Aneurysm Excision)

#### Rheumatic Heart Disease—Subacute Bacterial Endocarditis

**Case 24** Mrs T F an American housewife of 30 was first seen on September 3 1936 There were no cardiovascular symptoms She was pregnant at the third month and her physician requested evaluation of a systolic apical murmur discovered on routine physical examination

The patient had one attack of rheumatic fever at the age of 12

Physical Examination B P 108/80 Pulse 80 No irregularity A rough systolic murmur was heard over the apex transmitted well into the axilla The pulmonic second sound was accentuated Slight cardiac enlargement was present The electrocardiogram was normal

Clinical Diagnosis A Etiologic Rheumatic Inactive B Anatomic Slight cardiac enlargement Mitral insufficiency C Physiologic Normal sinus rhythm D Functional Classification Class 1 Therapeutic Classification Class B

The opinion was given that the pregnancy would not be complicated by any cardiac symptoms Routine examinations were advised (Chapter 15)

This patient was next seen on 9/16/38 Her pregnancy had been uneventful She was well until a month before the examination when she developed headaches weakness and slight dyspnea

Physical Examination Cardiac status unchanged The spleen was not palpable The blood count hemoglobin 90 per cent (Sahli); R B C 4 800 000 W B C 11 200 Blood culture negative

When re-examined one month later 10/16/38 the patient had lost eight pounds in weight Her complaints were weakness, palpitation and dyspnea The temperature was 99 3 F She was admitted to the hospital for study A week later the blood culture was positive for *Streptococcus viridans*

Discussion When the cause of this patient s symptoms was revealed by the positive blood culture she was ambulatory slightly overweight and appeared to be in good health The amount of cardiac damage resulting from one attack of rheumatic fever in childhood was slight and well healed She came through one pregnancy without complications was able to manage her household efficiently and took part in numerous outside activities

The development of subacute bacterial endocarditis in a young person of this type whose heart damage has been trivial and certainly compatible with a reasonable life expectancy is tragic and calls for heroic measures To give a hopeless prognosis on the basis of the blood culture to families of these patients is not an easy task particularly when the victims glow of health at the time is but slightly dimmed Newspaper and radio health talks that also bring to the fireside the news of wonderful advances in medical science have little to say regarding this disease However other well advertised scientific discoveries naturally lead relatives to expect that *surely something can be done*

Consequently when the *S viridans* was cultivated from the blood of this patient vaccines from the organism were prepared and injected into suit able donors in ascending doses in an attempt to procure immune blood Three robust donors were inoculated over the course of three weeks At the end of this period of preparation the blood serum of each agglutinated the *S viridans* in high titer During the patient s first month in the hos pital ordinary transfusions of 500 cc of whole blood had been given every week There were no reactions and the blood count was kept at 5 000 000 During the fifth week transfusions from the immune donors were begun When 100 cc of the first transfusion had been injected so severe a reaction developed that the transfusion had to be discontinued Five days later only 50 cc of the blood drawn from the second donor were given before a similar marked reaction took place The patient refused to take the next transfusion and the third donor was not used

The failure of this therapeutic procedure was discouraging and the severity of the reactions experienced by this patient makes me hesitate to recommend it In addition the method is time-consuming and expensive and usually it is difficult to get professional donors to submit to injections of vaccine

Recently Kilgore[189] recorded three similar failures in cases where the amount of immune blood given exceeded that in any of the previously reported cases while Howell Portes and Beverley are more hopeful [163] Nevertheless their patient died a week after the last transfusion The improvement was temporary following each injection of the immune blood and may have been due to increase in the blood count or the agglutinins for the infecting organisms The possibility also exists that the effect was purely psychic

In addition to injections of immune blood Lamb gave serum from an immunized horse and reports an unfavorable result [00] Other accounts in

literature dealing with immune hemotherapy in subacute bacterial endocarditis are not enthusiastic[163, 199]

The use of the immune donor however, appeals to the representatives of the family They view the doctor as a modern Galahad fighting the microbe with strange and intricate weapons and as a result they have much to tell abroad The morale of everyone connected with the case enjoys a temporary elevation, which is reflected at once in the patient's attitude This is my impression of the value of immune hemotherapy

During the second month of this patient's stay in the hospital the steady downward course was evident Transfusions of whole blood were *used frequently but in spite of them the blood count fell to 54 per cent* hemoglobin, 2,300 000 red blood cells by the end of the eighth week Occasional embolic phenomena were now in evidence in the extremities (tender nodes) the kidneys (hematuria, backache) and the spleen (tenderness enlargement) Weight was lost rapidly and the degree of toxemia increased

Toward the middle of the third month a greater degree of cardiac enlargement was noted the liver became enlarged and tender and edema of the feet dyspnea and slight cyanosis appeared While these signs of heart failure are unusual in the cases of subacute bacterial endocarditis they may be explained by the fact that the patient escaped major embolic accidents as the months went b In this event it is only natural to suppose that the myocardium would become the seat of sufficient structural damage to account for its failure[4] Therefore it is evident that we need no longer make use of that indefinite term toxemia to explain the myocardial weakness and failure that develop in the course of subacute bacterial endocarditis Sudden rupture of a valve the seat of a mycotic aneurysm may add an additional load that precipitates failure in some instances

In the presence of fever and such extensive myocardial injury it is little wonder that no response was obtained when the patient was digitalized Vitamin $B_1$ (thiamin chloride) and C (cevitamic acid) were given in large amounts by mouth and parenterally when edema appeared with no obvious result An increasing stupor was soon replaced by coma and the patient died at the beginning of the thirteenth week

A postmortem examination was not obtained in this case nor was it requested There are times when the physician finds it impossible to go to willing co operative families with a last request of this nature particularly if he knows from the close contact of weeks that this debt to science if paid will immeasurably increase the burden of grief

SUBACUTE BACTERIAL ENDOCARDITIS—INSIDIOUS ONSET AND LONG DURATION —AUTOPSY

**Case 25** W W a white male physician of 54 was admitted to the Philadelphia General Hospital on 11 17/31 complaining of fever and loss of weight

HISTORY Over a year before admission the patient was awakened by a sudden severe backache radiating toward the left sacro iliac joint The pain was partially relieved by aspirin and he was able to attend to all the duties of his practice At the end of a week he consulted an orthopedic specialist who following a roentgen ray examination made

a diagnosis of osteoarthritis of the acromioclavicular joints. A belt was prescribed which gave the patient some relief although the backache continued to be troublesome.

Headaches were then present every day and slight pallor was noticed by members of the family. Consequently the doctor visited another colleague and had his glasses changed. He also stopped by the hospital and had a blood count. A moderate secondary anemia was found. A vacation away from home was the prescription, anemia and overwork the diagnosis.

A month at the seashore was of little value. On his return to the city a loss of five pounds in weight was noted. There was a slight cough and an evening temperature as high as 100° F. A chest specialist was consulted who found evidences of tuberculous infiltration at both apices and sent the patient home to bed. Inquiry revealed that two brothers and one sister had died of tuberculosis.

During the next four months at home little improvement was observed. The temperature continued, the anemia increased and the patient lost 26 pounds in weight.

When admitted to the hospital the physical examination revealed pallor, a temperature of 100° F, a pulse of 80, and the blood pressure measured 96/84. There was dulness on percussion over both apices. No rales were elicited. The apex beat was in the sixth interspace just outside the midclavicular line and there was a harsh apical systolic murmur transmitted into the left axilla. The fingers were clubbed.

LABORATORY DATA. Blood count: hemoglobin 60 per cent (Sahli), R.B.C. 3,000,000, W.B.C. 12,000. Wassermann reaction negative. Six sputum examinations were negative.

At the end of the first week the blood culture was reported positive for *S. viridans*. The same day petechiae appeared in the conjunctivae and the spleen was palpable for the first time.

CLINICAL DIAGNOSIS. A. Etiologic: Subacute bacterial endocarditis, *S. viridans*. B. Anatomic: Slight cardiac enlargement. Mitral regurgitation. C. Physiologic: Normal sinus rhythm. D. Functional Classification: Class I. Therapeutic Classification: Class E.

The course was slowly downhill and the patient died 12/14/31, *15 months after the* onset of symptoms.

AUTOPSY. The heart weighed 300 Gm. The pulmonic valve was normal. The tricuspid valve showed a few vegetations about 1 to  mm. in diameter. The left side of the heart (Fig. 86) revealed extensive growth of vegetations on the free border of the mitral valve, *extending down on the chordae tendinae and on the left auricular wall. The* aortic valve was *normal. Both lungs revealed healed apical lesions of tuberculosis. The* spleen and kidneys showed multiple areas of infarction.

DISCUSSION. The history of the onset of this patient's illness is typical of the disease. Often vague joint pains and headaches occur as prodromal symptoms. Fever may develop during the early stages and disappear for long intervals, but it always recurs and once established seldom leaves the picture completely for any length of time.

This patient's story brings out the danger of viewing pain in a location that happens to be the official precinct of a specialist as a result of a disease process at this site. There is no doubt that a mild degree of osteoarthritis was present in the region indicated, but the local findings in this case did not explain the whole picture. The same observation holds true with regard to the headache. The patient was a physician, and for this reason his fellow practitioners took too much for granted when he consulted them in regard to a symptom. It was quite natural, when the history of tuberculosis in the family was revealed and the roentgen study showed bilateral apical shadows, that the expert on chest should feel that the patient's case was fully solved and advise him accordingly. We are all apt to develop "blind spots" for certain unexplained symptoms when we are either too tired or too hasty. Unfortunately, at a later date we may see

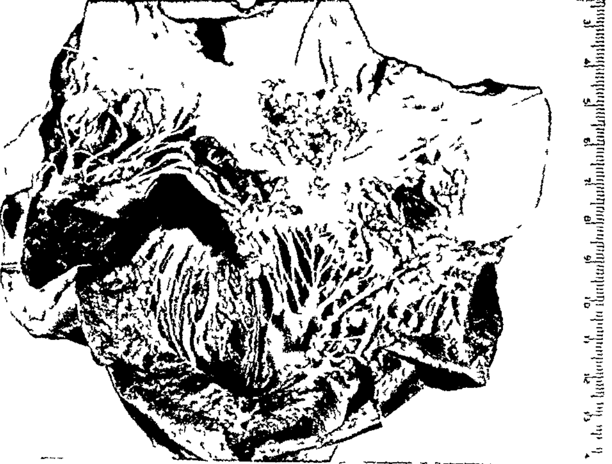


Fig 86 Subacute bacterial endocarditis Note the extensive growth of vegetations on the free border of the mitral valve the chordae tendineae and on the wall of the left auricle (Autopsy No 3 663 Philadelphia General Hospital)

these symptoms develop to embarrassing proportions The flower in full bloom may be readily named by a colleague when its species is difficult to recognize in the bud

When this patient was viewed as a whole the parts of the puzzle went into place very readily The embolic manifestations were recognized and the positive blood culture clinched the diagnosis The autopsy showed an extensive growth of vegetations not only on the heart valve but also on the auricular wall The apical lesions of tuberculosis were found but they were completely healed The areas of infarction in the kidneys suggested another cause for the backache that ushered in the illness

### Subacute Bacterial Endocarditis Accompanied by Multiple Embolic Episodes—Autopsy

**Case 26** R F K a male clerk of 31 was admitted to a surgical service at the Woman s College Hospital on January 1935 complaining of sudden pain in the abdomen

History For two months following a dental extraction the patient had complained of weakness vertigo and headache and was unable to work for the week before admission because of increase in the severity of the symptoms The past history showed attacks of rheumatic fever at the ages of 11 and 13

Physical Examination T 101 F P 110 B P 150/40 Pallor Corrigan pulse There was cardiac enlargement to the anterior axillary line The heart rhythm as regular There was an accentuation of the first heart sound at the apex and in the same area a diastolic thrill was palpable Presystolic and diastolic murmurs were heard in the mitral area Along the left sternal border there was a loud diastolic murmur The spleen was palpable and tender The fingers showed marked clubbing

Laboratory Data Blood cultures 1 5 colonies of *Streptococcus viridans* per cc of blood Wassermann reaction negative Blood count hemoglobin 55 per cent (Sahli) R B C 3 200 000 W B C 14 000 The urine showed 10 R B C per H P F

Clinical Diagnosis A Etiologic Rheumatic Subacute bacterial endocarditis *S viridans* B Anatomic Aortic insufficiency Mitral stenosis Mitral insufficiency C Physiologic Normal sinus rhythm D Functional Classification Class 3 Therapeutic Classification Class E

Discussion Needless to say this patient did not require surgical treatment Viewing the history subsequent to the extraction of three teeth we cannot state that the development of subacute bacterial endocarditis was merely a coincidence I have many times seen the *S viridans* gain a foothold in a previously damaged endocardium following an ill advised tonsillectomy or dental extraction This patient had three teeth removed because of abscesses that were demonstrated on roentgen examination He was *advised to have this done because of heart trouble* A local anesthetic was injected which may have forced infected material lurking about the margins and roots of the teeth deep into the capillaries and given it a good start on its way to the heart valves

In all patients who have damaged hearts particularly the younger ones where rheumatic and congenital types predominate dental extractions should not be freely advised or carelessly undertaken A complete physical examination should first be carried out Anemia and dietary deficiencies brought to light should first be explained and then corrected by iron

vitamins and a proper diet This may help to increase those forces of resistance that we so often refer to in our daily practice

When the patient is ready to have the dental operation the surgeon should first give the teeth a thorough cleansing to remove the contaminants from the field Extractions should then be carried out at sensible intervals After each extraction a thorough cleaning of the socket is advisable and packing should be avoided if possible All patients known to have heart lesions should be referred first to their physician by the dentist before any operative work is begun in order that responsibility may be shared and all the protective measures employed

It seems likely that *S viridans* invades the blood stream of normal individuals many times during a lifetime This fact may often be demonstrated by the use of special technic [08, 311] I am not convinced that the endothelial structures are more permeable in late winter and spring and less permeable in summer and autumn and that we should confine our operations to these favorable seasons When we advise operations on teeth and tonsils at any season in patients known to have heart disease the use of sulfanilamide or one of its derivatives before and after the operation is worthy of trial to combat the transient bacteremia that often occurs

TABLE V

SULFANILAMIDE DOSAGE

The Amounts of Sulfanilamide Necessary to Establish Effective Blood Levels (10 to 15 Milligrams Per Cent) Quickly in Patients Ill with Severe Hemolytic Streptococcal Meningococcal Gonococcal Pneumococcal or Welch Bacillary Infections

| Weight of Patient | | Initial Dose *Per Os* | | Maintenance Dose *Per Os* q 4 Hours (Day and Night) | | Total Dose First 24 Hours | | Total Daily Dose Bicarbonate of Soda | |
|---|---|---|---|---|---|---|---|---|---|
| Kilos | Pounds | Grams | Grains | Grams | Grains | Grams per Kilo | Grains per Pound | Grams | Grains |
| 70 | 150 | 4 8 | 80 | 1 2 | 20 | 0 15 | 1 2 | 3 6 | 60 |
| 60 | 125 | 4 2 | 70 | 0 9 | 15 | 0 15 | 1 2 | 3 0 | 50 |
| 45 | 100 | 3 6 | 60 | 0 9 | 15 | 0 18 | 1 3 | 3 0 | 50 |
| 35 | 75 | 3 6 | 60 | 0 9 | 15 | 0 23 | 1 8 | 3 0 | 50 |
| 23 | 50 | 3 0 | 50 | 0 6 | 10 | 0 26 | 2 0 | 1 8 | 30 |
| 11 | 25 | 1 8 | 30 | 0 3 | 5 | 0 30 | 2 2 | 0 9 | 15 |

From Long and Bliss Clinical Use of Sulfanilamide and Sulfapyridine and Allied Compounds —Reprinted by permission Macmillan Co N Y

When subacute bacterial endocarditis develops however the use of sulfanilamide derivatives alone have no effect on the course of the disease This patient was the first under my continuous care where sulfanilamide was used in an attempt to rout the invasion of the *S viridans* Very slightly lower doses were given than have since been recommended by Long and Bliss [36] *in Table V An initial dose of 4 0 Gm (60 grains)* of sulfanilamide was administered and this was followed by 1 0 Gm (15 grains) doses

every four hours accompanied by a similar amount of sodium bicarbonate No untoward effect was noticed During this week there were many variations in the temperature but it is impossible to state whether they were due to the drug since similar remissions occurred in the absence of sulfanilamide Blood cultures were negative during the period in which sulfanilamide was given but were positive again when the drug was discontinued demonstrating the failure of the chemical to penetrate the depth of the lesions on the heart valves

There are comparatively few reports in literature of large series of cases of subacute bacterial endocarditis treated with sulfanilamide Isolated instances of recovery have been reported by Hussey[166] Major and Leger[2 4] One patient reported by the last observer was cured by combined sulfanilamide and neoprontosil therapy but died of congestive failure 29 days later Autopsy showed damage of the aortic and mitral valves but cultures of removed segments were sterile While the diagnosis in other reported cases of cure may be open to doubt the patient reported by Major and Leger had a history of rheumatic fever and old mitral and aortic valvular lesions petechiae splinter hemorrhages tender fingers fever enlargement of the spleen and three blood cultures positive for *S viridans* Necropsy showed the evidence of a recent endocarditis in the stage of healing and repair The question of course arises whether or not cure was produced in this single instance by the use of the drug or whether it was another example of spontaneous recovery We can be hopeful that it was an example of the efficacy of the early use of sulfanilamide before the bacteria in the valve lesions became inaccessible since the time of the institution of sulfanilamide therapy in these cases appears to be a most important factor

Spink and Crago[3 0] in a detailed evaluation of sulfanilamide therapy in 12 cases of subacute bacterial endocarditis report a bactericidal effect that was temporary and depended on continued use of the drug in 10 of the 12 instances They conclude that the value of sulfanilamide is doubtful Long and Bliss[36] have observed the effects of sulfanilamide in more than 60 cases of subacute bacterial endocarditis They report five cures in four of these patients the infection was engrafted on a congenital lesion and in the fifth case there was a rheumatic background

I still give sulfanilamide to every patient under my care who is suffering from subacute bacterial endocarditis If the drug is well borne I continue it in sufficient dosage to maintain the concentration of 10 to 15 mg per cent I have always used sulfanilamide by mouth The only tests of efficacy of this treatment are the blood cultures *

Sulfapyridine gives the same results in subacute bacterial endocarditis as sulfanilamide Bacterial growth disappears from the blood culture as soon as the drug is administered In some cases this is attended by a fall

* All blood cultures for *Streptococcus viridans* should be incubated for at least 10 days before a negative report is made This precaution is especially advisable when a chemotherapeutic agent is present in the blood sample

in the temperature When the drug is withdrawn and in some cases while it is still being given all the clinical features of the disease return in much the same intensity as before Long and Bliss advise the use of a total daily dose of 0 1 Gm per kilogram of body weight in patients weighing up to 60 kilograms The dose should be divided and given at intervals of six hours

Kelson and White[183] have recently reported their experience in using sulfapyridine combined with heparin They call attention to the fact that the streptococci lie near the periphery of the vegetations embedded in fibrin and platelets that act as an efficient wall preventing the contact of leukocytes with the bacteria Heparin an anticoagulant is used to limit the growth of the vegetations to encourage fibroblastic invasion of the area and to prevent if possible embolic accidents that invariably follow the extensive thrombus formation

Technic of the method proposed by Kelson and White is as follows dissolve 10 cc of heparin (10 000 units) in 500 cc of physiologic saline and administer by uninterrupted intravenous drip day and night for 14 days The flow is regulated to maintain venous clotting time (normally below 20 minutes) at approximately one hour Clotting time must be determined before treatment, twice during the first day and then daily until the heparin is discontinued The sulfapyridine in suitable amounts is started (Table X) *from four to seven days before the heparin* and continued during the heparin treatment and for one week following its withdrawal Blood transfusions are given if the blood count falls below 3 500 000 red blood cells To increase the efficiency of fibrous repair the patient is saturated with vitamin C It is well to give 200 mg of cevitamic acid by mouth four times a day for three days and then continue it in daily doses of 100 mg

Three of the first six patients reported by Kelson and White who were able to take the heparin for more than a week showed striking improvement and have remained free of evidences of the disease for periods of ten weeks 18 weeks and four weeks respectively, after discontinuing treatment These afebrile intervals are much longer than usually occur in control cases

Certain dangers attend the use of heparin but these must always be viewed in the light of the hopeless prognosis that the disease invariably carries with it Two patients of this series had toxic reactions to the heparin death resulting in one case in 17 hours Another danger that must be considered with the use of heparin especially in a disease like subacute bacterial endocarditis is fatal hemorrhage due to interference with clotting following embolic episodes Friedman and his associates[108] emphasize this danger following heparin in their report of a single case where a fatal cerebral hemorrhage terminated the picture ten days after heparin was administered These authors also believe that some danger attends the sudden liberation of large quantities of bacteria when the vegetations are broken up

The final opinion in regard to the effect of heparin sulfapyridine therapy in subacute bacterial endocarditis must await the result of further careful clinical and laboratory studies Additional animal experiments must be carried out to prove the action of heparin in the concentrations recommended Does it actually prevent the deposition of platelets and fibrin on the valves as claimed? Will this action allow Nature to gain the upper hand and produce firm healing in these areas? These answers we hope



FIG 87 Subacute bacterial endocarditis Infarct of spleen following embolism

will soon be obtained Meanwhile every effort should be directed toward early diagnosis when the vegetations are fresh and small for only under these circumstances can we hope that this plan of attack will have the success that the early case reports have led us to expect

For the present it must not be assumed that a cure has been found My personal experience with heparin in subacute bacterial endocarditis has been most discouraging The dangers in my opinion are prohibitive even when we consider the nature of the disease this therapy has been fashioned to combat and they should always be explained to the patient s family before the treatment is begun Heparin should only be used in the

hospital where its effects can be constantly observed and the results appraised after an adequate follow up period

In the presentation of this new plan of chemotherapeutic attack with its many variations we have digressed for the moment from the case under discussion Embolic manifestations showed the same tendency to occur during the second week in fact were more frequent after sulfanilamide was begun On the tenth hospital day the patient died suddenly from what we considered was a massive cerebral embolism

The autopsy showed old rheumatic lesions of the mitral and aortic valves with large masses of vegetations in both locations A large splenic infarct (Fig 87) explained the abdominal pain that was present on admission Both kidneys showed the numerous scars and petechiae (see Fig 85) that are so characteristic of subacute bacterial endocarditis

## Streptococcus Viridans Septicemia (Subacute Bacterial Endocarditis?)—Attempted Cure by Excision of Arteriovenous Aneurysm

**Case 27** T B a negro laborer of 36 was admitted to the Philadelphia General Hospital on 9/9 38 complaining of pain in the joints chills and fever

History The patient was well until a month before admission when he developed a chill following considerable out of door exertion *This was followed the next day by joint pain and fever The fever continued until the time of admission to the hospital a month later During this time he lost* [illegible]0 *pounds in weight*

Past Medical History A lesion suggestive of chancre was present [illegible]0 years prior to admission There was a gunshot wound of the left thigh *nine years before admission*

Physical Examination T 100 F B P 110/45 The area of cardiac dulness was increased in all diameters The apex beat was palpable in the fifth interspace *13 cm* to *the left of the midclavicular line No thrills were present* The pulse was *rapid* Corrigan in type and the rhythm was regular There was a loud blowing systolic murmur heard over the entire precordium louder in the upright position and a diastolic murmur was heard over the base of the heart The pulmonic second sound was accentuated The liver edge was firm and smooth and distinctly palpable under the right costal margin The spleen was palpable A thrill was felt over the femoral canal [illegible] cm below Poupart's ligament in an area measuring 16 sq cm On auscultation over the point of maximum intensity of the thrill a deafening bruit was heard that could also be detected along the proximal vessels to the umbilicus and along the distal vessels to the ankle Digital pressure over the point of greatest intensity caused both bruit and thrill to disappear Reflexes were normal except the patellar and Achilles on the left side which were absent Pulsations of the popliteal posterior tibial and dorsalis pedis were readily felt on the right but were barely palpable on the left side No varicosities or edema were noted in either thigh leg or foot No clubbing of the fingers or toes was present The skin temperature was the same on both sides The blood pressure in the right arm measured [illegible] in the left arm 93/30, in the right leg [illegible]/75 and in the left leg 85/50 The venous pressure varied from 115 mm to [illegible] mm

Laboratory Data A blood culture taken on the day of admission showed [illegible] colonies of *Streptococcus viridans* per cc of blood A roentgen ray study of the heart showed enlargement in the transverse diameters There was hypertrophy of the left ventricle and some widening of the aorta (Fig 88A) A film taken after compression of the arteriovenous aneurysm (Fig 88B) showed the heart to be slightly smaller and [illegible] Wassermann negative

Clinical Diagnosis Arteriovenous aneurysm Probable focus for *S viridans* septicemia Cardiac hypertrophy with aortic regurgitation etiologic type unknown

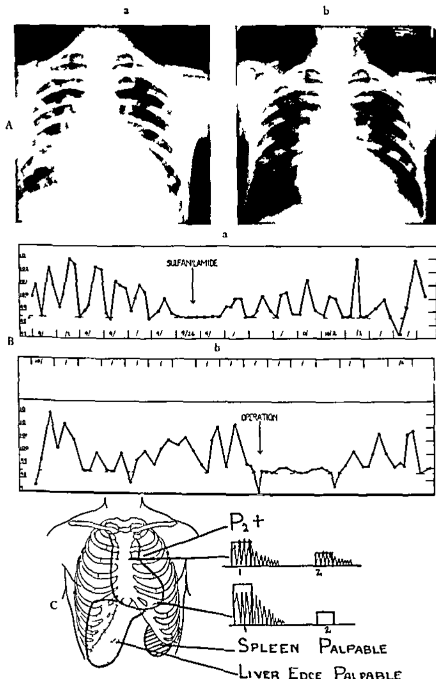



FIG. 88. A. Roentgenograms showing heart size (a) before compression and (b) after compression of the arteriovenous communication.
B. Temperature chart showing (a) effect of sulfanilamide therapy and (b) effect of operation.
C. Chart illustrating physical findings.

Discussion Following admission the fever persisted, and the anemia became more pronounced A second blood culture again showed *S viridans*, so it was evident that a focus was present and active Viewing the degree of cardiac damage it would appear that the blood stream infection, if primary in the aneurysmal varix had already reached the heart valves Continued observations however, failed to disclose any change in the character of the murmurs, consequently operation to remove the arterio venous aneurysm was considered If syphilitic heart disease were present with valves undamaged by the blood stream infection, removal of the infected varix offered the possibility of cure On the other hand if endo cardial involvement already existed the operation could do no harm and might benefit the patient to the extent of removing the strain of the arteriovenous aneurysm on the already damaged heart

The continued absence of embolic manifestations was a point against the involvement of the mitral or aortic valves or the endocardium of the left side of the heart Evidence of damage to the femoral artery and vein and of the re establishment of a subsequent fistula was sufficient to make us suspect that the point of origin of the septicemia might be in this area Sulfanilamide was tried from 9/21/38 to 9/26/38 (Fig 88B) and it suc ceeded in producing a transient fall in the temperature to normal and a temporary reduction in the number of *S viridans* colonies in the blood stream Since a marked increase in the degree of anemia was noted blood tranfusions were given When sulfanilamide was discontinued the tempera ture returned and the number of colonies per cc in the blood culture rose to the former level

After repeated trials, the status of the collateral circulation was found to be excellent consequently surgical removal of the arteriovenous aneu rysm was decided upon A drawing (Fig 89) made at the time of the operation shows the dissection of the aneurysmal communication between the left femoral artery and vein in this patient After ligation the entire aneurysmal sac was removed Examination of the specimen showed on the arterial side of the communicating channel a ring of dark red friable vegetations No vegetations were present on the venous side of the orifice Microscopic examination of the sections using hematoxylin and eosin stain showed small clumps of dark blue cocci like bodies interpreted as being bacteria similar to the bacterial masses commonly seen on the heart valves in cases of subacute bacterial endocarditis

After operation the leg showed little change There was a slight decrease in the temperature of the operated side and a very slight swelling The patient s temperature (Fig 88B) promptly fell to normal following the operation The cardiac rate likewise dropped and clinical improvement was evident For nearly a week we were of the opinion that cure of the blood stream infection following the excision of the focus containing the vegetations had taken place However we were mistaken At the end of the first postoperative week the blood culture became positive and the temperature again began to show a gradual elevation On transfer back

B

A

[illegible]

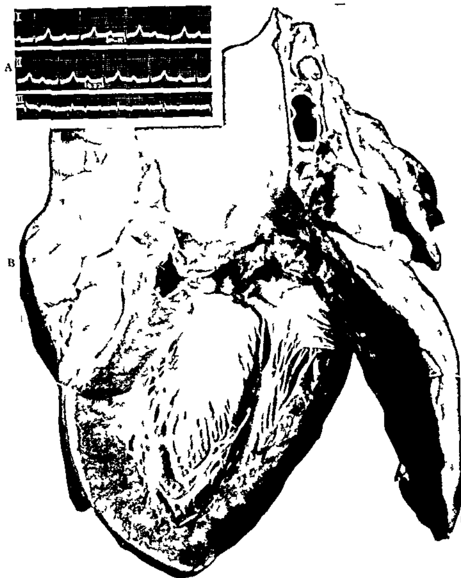



Fig 90 A The electrocardiogram Note prolongation of the P R Intervals to 0 3 second

B Subacute bacterial endocarditis superimposed upon calcific aortic stenosis The margins of the leaflets are fused thickened and calcified Note the extreme hypertrophy of the left ventricle and the smooth aortic wall above the valve (Autopsy No 27 911 *Philadelphia General Hospital*)

to the medical ward sulfapyridine was begun but the effect was the same as observed following sulfanilamide The temperature fell to normal, and the blood cultures were reported sterile from 11/16/38 to 11/30/38 On the latter date increasing dyspnea cyanosis and edema ushered in a rather sudden attack of congestive failure, and the patient died Autopsy was refused

Although we were unable to duplicate the success of Hamman and Rienhoff[146] and obtain a radical cure in this patient, we believe that if we had seen him earlier and had operated on him at once, the story might have had a different ending This case illustrates a type of heart disease rarely encountered where cure may be possible by excision of an infected arteriovenous communication

While we are discussing the value of operative procedures in subacute bacterial endocarditis attention must be called to the recommendation of splenectomy made by Riesman [316] When the spleen is large the patient's nutritional state good and congestive failure absent this operation imposes little additional risk The spleen is involved early in the course of the disease, consequently its role in feeding the blood stream may be an important one Viewed in this light its removal seems just as logical as the extirpation of the pelvic organs to prevent continual infections in gonococcal endocarditis or the removal of the arteriovenous aneurysm in the case just presented

The incidence of subacute bacterial endocarditis in patients with uncomplicated patent ductus arteriosus is high In Abbott's series of 92 cases death was caused by subacute bacterial endocarditis in 21 Graybiel Strieder and Boyer[140] were the first to propose surgical ligation of the ductus in these cases in an attempt to shut off the pathway to the blood stream They point out that the infection begins in relation to the pulmonary orifice of the ductus extending often to the pulmonary valves but rarely to the aorta Hence if an early diagnosis is made in cases of patent ductus arteriosus which is not impossible if they are constantly under observation surgical intervention must be considered Ligation has been successfully carried out by Gross (page 339) and seems justified in selected instances in an attempt to bar the bacteria and the vegetations from the blood stream in the hope that natural forces may be effective in healing the areas

### Calcific Aortic Stenosis—Superimposed Subacute Bacterial Endocarditis—Autopsy

**Case 28** W H a German cabinet maker of 49 was admitted to the Philadelphia General Hospital on 2/7/34 complaining of cough and dyspnea

History The patient was in fairly good health until two months before admission when he noticed chest pain that increased on exertion and radiated to the left arm Dyspnea was present A buzzing was felt in the chest when in bed at night Two weeks before admission edema of the ankles appeared for the first time this increased in severity until the day of admission

The patient denied venereal disease There was no past history of rheumatic infection

Physical Examination B P 110/90 T 101 F P 90 Petechiae present on the

conjunctivae The apex beat was in the sixth interspace 1 cm from the midsternal line A marked thrill was palpable over the second interspace to the right of the sternum A harsh systolic murmur was present over the same area transmitted to the vessels of the neck A systolic murmur was heard over the margin of the cardiac apex Rales were heard over both lung bases The liver edge was tender and palpable a hand's breadth below the right costal margin The spleen was not palpable No clubbing

LABORATORY DATA Blood culture negative Wassermann four plus Kahn negative R B C 3 ?00 000 W B C 5 600 hemoglobin 65 per cent (Sahli) P 76 L 4

Electrocardiogram prolongation of the P R intervals (First stage heart block) (Fig 90A)

Roentgenogram dilated aorta and cardiac enlargement (probably luetic)

CLINICAL DIAGNOSIS A Etiologic Rheumatic? Arteriosclerosis? Syphilis? B Anatomic Cardiac enlargement Aortic stenosis Relative mitral insufficiency C Physiologic First stage heart block Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class F

COURSE Congestive failure increased in spite of the usual therapeutic measures The blood cultures were negative although the patient had an irregular temperature for two weeks before death

AUTOPSY The heart weighed 660 Gm There was hypertrophy of the left ventricle The mitral valve was normal The aortic valve was entirely destroyed and replaced by calcium Superimposed upon this were firm masses of thrombi some of which showed calcification The end of a slate pencil could scarcely be pushed through the unopened aortic valve The aorta above the valve was smooth and conspicuously free of any pathologic change in contrast to the marked calcific lesion of the valve (Fig 90B)

**Discussion** The aortic regurgitation and the positive Wassermann reaction in a man of 46 first suggested syphilis as the etiology However in the presence of the signs of subacute bacterial endocarditis the possibility that syphilis was the only etiologic factor diminished While bacterial endocarditis may be superimposed on syphilitic aortitis and valvulitis as the recent studies of Martin and Adams [8] indicate nevertheless it is a rare event The diagnosis should always be considered however in the presence of sepsis of undetermined origin a positive Wassermann reaction evidence of aortic insufficiency and a negative rheumatic history In this patient the harsh systolic murmur and thrill discovered over the aortic area in the absence of aneurysmal dilatation favor the diagnosis of a stenotic (rheumatic) lesion The calcific type of aortic stenosis has been discussed elsewhere (page 127)

The petechiae and the course of the fever in this patient were characteristic of subacute bacterial endocarditis even in the absence of a positive blood culture and also accounted for the positive Wassermann reaction *This occurrence of biologic false positive Wassermann reactions in the presence of subacute bacterial endocarditis is not unusual* Consequently it is not surprising that neoarsphenamine therapy has been suggested for these patients Stokes however opposes its use [7] Autopsy in this case proved the absence of syphilis and verified the correctness of this interpretation of the serology

The rapidity of the organization and calcification of the thrombi in this case in the absence of any treatment directed toward the process shows the natural tendency of the disease to heal

While the bacteriologist who isolates the *S viridans* from the blood *stream may refer to this organism as an invader of low virulence,* yet we are beaten as soon as he makes the diagnosis The mechanical features of the infection in most instances close the picture Masses of thrombi build up break off and are carried to vital centers by the blood stream In this patient it is hard to understand why the organism would pick out such a cardiac desert area as a totally calcified valve for implantation The valve it is true was the seat of previous damage but certainly in its calcified state it contained less blood supply than other lesions

The healing tendencies in this disease are evident in this desperate and almost successful attempt to organize and calcify the thrombi However the increase in the obstruction that this produced almost completely closed the valvular orifice and precipitated cardiac failure The absence of bacteria in many blood cultures is proof of the fact that the healing process was *gaining the upper hand If in the future we can keep the blood stream* sterile in subacute bacterial endocarditis and prevent large thrombotic masses from forming on the valve sites long enough for fibrous tissue to invade and organize the mass already present we may succeed in conquering this infection

# 6

# SYPHILITIC CARDIOVASCULAR DISEASE

The same medicine will both harm and cure me—OVID
*Tristia* Bk ii 1 20

The incidence of cardiovascular syphilis in any country today is an index of the intelligence of its population in general and of the ability and foresight of its medical profession in particular While rheumatic infection remains one of the grave problems of modern civilization syphilitic heart disease can be greatly minimized by the recognition and adequate treatment of early lues Considering the high mortality rate among patients with advanced cardiovascular syphilis it is small wonder that a nation wide intensive campaign was launched recently to educate the public and bring the early cases under competent medical supervision The careless attitude of the average private physician in regard to the treatment of syphilis has been responsible for organized efforts on the part of the governments of many countries to combat the disease Since a full understanding of the anatomic and pathologic aspects of syphilitic involvement of the heart and great vessels is a necessary prelude to intelligent management a brief review of these fundamental principles will precede the discussion of therapy

## THE LESION

**Chancre** The primary lesion of syphilis is the chancre Accumulative evidence points to the fact that the blood stream is invaded by the *Treponema pallidum* before the appearance of this primary lesion It may be possible in many cases that the spirochete has already reached the aorta before clinical manifestations of syphilitic infection are noted When a chancre is in evidence local medical or surgical measures directed toward the initial lesion are of no avail since both blood stream and lymphatic system have transported the spirochete far beyond the reach of regional medication Many spirochetes invade nearby lymph nodes and are killed by the resistance forces of the body Others penetrate deep into the lymphatic channels finally reaching the thoracic duct and the venous blood stream During the initial shower of spirochetes many penetrate as far as the lymph nodes in the thorax and from here by lymph movement reach the vasa vasorum in the aorta

**Obliterative Endarteritis** In these small vessels within the artery wall an initial obliterative endarteritis develops which may slowly spread

to the whole ascending portion of the aorta The seriousness of this destructive process can readily be realized when we recall that the cusps of the aortic valve are attached in this area When this section of the aorta becomes the seat of scar formation the valve is drawn backward and regurgitation develops It is important to note that the damage takes place without stenosis which distinguishes it from the rheumatic type in which adhesions develop between neighboring cusps, obstructing the flow of blood and at the same time interfering with valvular function

**Aneurysm** If the spirochetal invasion in the aortic wall is unchecked it plays havoc with the muscular media Degeneration of this coat is reflected in the pallor and wrinkling of the intima The whole aortic wall becomes weakened by the destruction of this vital tissue, eventually dilates and saccular aneurysms result If the process of dilatation widens the aortic valve and causes regurgitation the heart will be affected, with the development of cardiac hypertrophy However if regurgitation does not occur and the coronary ostia escape an aneurysm of the aorta usually has no effect on cardiac size or function It can, of course cause symptoms by pressure on any of the surrounding structures depending on the part of the arch involved and the direction in which the sacculation points For example the aneurysm may press on the trachea and bronchi with the appearance of the typical brassy cough Adhesions to the trachea may give rise to a tracheal tug (Oliver s sign) while compression of a bronchus if long continued will result in atelectasis If the aneurysm points posteriorly it may erode the vertebrae and cause severe pain The erosion on the other hand, may be in an anterior direction in which event a pulsating tumor will appear on the anterior chest wall (Fig 91) Aphonia appears when the recurrent laryngeal nerve is involved in the process Many times if aneurysms do not press on the structures above mentioned and do not cause erosion they may reach a large size without attracting attention

If aortic dilatation is moderate and regurgitation develops the pressure on the weakened arch is relieved and no aneurysm appears However the situation is still far from a happy one since cardiac hypertrophy occurs and the patient will eventually succumb to congestive failure

**The mouths of the coronary arteries** are situated in a bad neighbor hood as far as the syphilitic patient is concerned, and may be invaded early in the disease in which event serious symptoms occur Angina appears and with it the likelihood of sudden death at any time Willius[406 410] has shown that the coronary arteries occasionally rise above the level of the sinuses of Valsalva and in this location they stand a much greater chance of involvement in a syphilitic process than when they take their origin from the aorta at a lower level The effect of a syphilitic involvement of the coronaries differs in no way as far as the patient s symptoms are concerned from an arteriosclerotic process An anoxemia is produced in the heart muscle and this is attended by pain In syphilis the coronary arteries are generally free of involvement along their course which may

account for the infrequency of thrombosis Coronary occlusions following arteriosclerotic heart disease are more common during the winter months while those that complicate syphilitic aortitis show no seasonal variation Pean and Mills [3] believe this is a result of the steady progression of the syphilitic lesion

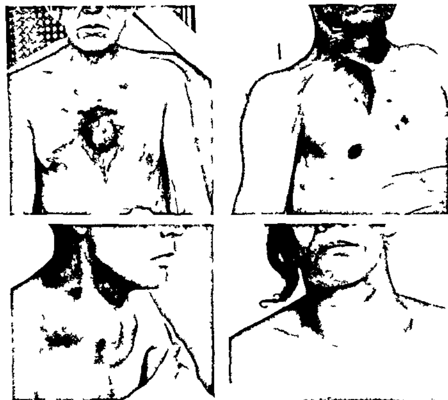


FIG 91 Aneurysms of the aorta S ellings of this type are at times confused with other tumors of the chest wall

The heart itself is seldom invaded by the spirochete If secondary changes are discovered in the heart muscle in syphilis they are usually produced by the narrowing of the coronary mouths in the aorta or result from a complicating arteriosclerosis Gummas of the heart producing heart block or other symptoms, are rare Von Haam and Ogden[143] collected reports of only seven cases from the literature between 1845 and 1935 In a study of 5213 autopsies of their own they reported gummatous lesions of the heart in only three instances

From this description it is evident that the earlier the cardiac invasion is detected the better will be the result of specific therapy However this

cannot be accomplished unless we suspect the presence of the infection The constantly alert physician will report a much higher incidence of cardiovascular syphilis in his practice because his suspicions are aroused by small details in the history and physical examination If instead of dismissing these small departures from normal he follows them with determination until positive proof is obtained either by further study from a confession from the laboratory or by a consultation cardiovascular complications of middle life may be largely prevented The student makes the mistake of regarding the aneurysm or the cor bovinum resulting from aortic regurgitation seen at autopsy as typical evidence of cardiovascular syphilis He returns to the wards to seek them clinically and later in his practice as a result of a lack of knowledge fails to recognize the earlier manifestations

## SIGNS

At this point the question properly arises What are the early signs of cardiovascular syphilis? The different criteria in use in various clinics and the lack of uniformity of opinion even among experts in the matter serve to confuse the practitioner We must acknowledge at the start that this diagnosis is most difficult and the criteria are far from settled However the careful physician should not be discouraged for here is a clinical problem that should challenge his skill and arouse his interest

**Aortic Second Sound** Unless the syphilitic process involves the coronary mouths and causes aortic regurgitation or weakening of the aortic wall its presence is apt to elude the physician who does not always keep the possibility in mind Certainly the scars in the first part of the aorta which the pathologist so triumphantly points to at autopsy can furnish him no clue at the bedside The higher up in the arch these scars are situated the greater will be the clinical silence they maintain However something can still be said concerning the character of the aortic second sound that accompanies this structural alteration Its tambour or drum like quality in the presence of syphilis may reflect a tissue change progressing in the aorta as a whole If we keep in mind the fact that hypertension or arteriosclerosis produce structural alterations, and consequently a similar change in the tone of the aortic second sound a tambour quality of the aortic second sound should serve to sharpen our suspicion It is not in itself diagnostic

**The systolic aortic murmur** that may be heard at the time of early syphilitic invasion is a less certain sign It too may accompany arteriosclerosis but at the same time it can also serve as a building stone in our diagnosis of early syphilitic aortitis

**Fluoroscopic Examination** If the patient who has these indefinite signs on physical examination is examined fluoroscopically suspicion may be strengthened The aorta in early syphilis is slightly wider and more dense than normal (page 43) and in addition there may be noted a

slightly increased pulsation These changes in my opinion are most suggestive and valuable when reported by an observer who has had considerable experience with the fluoroscopic method Even then they do not establish the diagnosis beyond doubt but only forge another link of similar strength in the chain of evidence

**Other systems of the body** may contribute more clear cut evidence of syphilis if the physical examination is carried beyond the region of the heart For example the presence of signs of neurosyphilis should be sought since *this form of involvement often co exists in the same patient* In the Co-operative Clinical Studies in 191 cases of uncomplicated syphilitic aortitis in which lumbar punctures were made within a month of the detection of aortitis 49 per cent showed unquestionable spinal fluid abnormalities Consequently a spinal fluid examination is an indispensable part of the examination of every patient who has cardiovascular syphilis

**Unequal Simultaneous Blood Pressure** Herzog[1 6] states that the diagnosis of syphilitic aortitis should be suspected when the blood pressures taken in both arms simultaneously are shown to be unequal To accomplish this two blood pressure cuffs are needed These are connected to the manometer by means of a Y tube The use of a symballophone enables the observer to detect the exact time of the appearance of the pulse beats in each arm

We cannot depend on any abnormalities of the electrocardiogram to diagnose syphilis Heart block occurs in association with sclerotic or coronary disease but is rarely caused by syphilis Auricular fibrillation is very rarely present as a complication of syphilitic aortitis a fact that may be valuable at times in ruling out syphilis in doubtful cases

## SYMPTOMS

The symptoms that are encountered in syphilitic aortitis are few and these are by no means characteristic The fact that they sometimes tend to be sudden in onset however should arouse suspicion The abrupt appearance of the signs of circulatory failure dyspnea cough edema substernal pain particularly in young patients may be significant The dyspnea may remain as a nocturnal occurrence while other signs of failure may progress These however are late manifestations and usually come on at a time when treatment offers less permanent benefit

Reports from the various clinics show that serologic tests in patients with cardiovascular syphilis are positive in 75 to 80 per cent of the cases[65 4 6] This figure however is much less when previous although inadequate treatment has been given[66] Consequently while a negative Wassermann test does not rule out syphilis (page 56) its routine use will serve to bring under scrutiny a larger group of patients before damage is evident in the cardiovascular system at a stage when properly direct treatment may prevent its development

## PROBLEM OF EARLY DIAGNOSIS

In the light of this review, it is evident that the detection of cardiovascular syphilis is quite easy if it is advanced, but difficult if not impossible *in its earlier stages* The earlier the diagnosis, the greater will be the success of the treatment from the cardiovascular standpoint No clue that can be obtained from a complete history, a thorough physical examination including neurologic survey and laboratory *study (roentgen ray fluoroscopy* electrocardiogram serologic tests of blood, and a complete spinal fluid examination including cell count globulin estimation, colloidal curve and quantitative Wassermann test) should be omitted Cardiovascular syphilis should then be diagnosed in from 5 to 25 per cent of the cases of heart disease in the practice of the average physician, depending of course on his location If his figures are less than the minimum stated above, he is probably missing the diagnosis in some instances

Welty[358] has recently analyzed the incidence of cardiovascular syphilis in the records of 15 000 consecutive autopsies at the Philadelphia General Hospital In 1 040 cases (6 93 per cent) cardiovascular syphilis was diagnosed Aneurysm was present in 192 patients aortic insufficiency in 216 and simple aortitis was found in the remaining number[63] Males predominated in this series (74 per cent) while 68 per cent were negroes When *these cases are divided into five groups of 300 autopsies each a decreasing* incidence of cardiovascular syphilis is seen (Table VI)

TABLE VI
DECREASING INCIDENCE OF CARDIOVASCULAR SYPHILIS AT THE PHILADELPHIA GENERAL HOSPITAL (After Welty)

| Years | Number of Cases | Incidence | Ratio |
|---|---|---|---|
| 1927 to 1930 | 276 | 92 per 1000 | 1 per 11 deaths |
| 1930 to 1932 | 231 | 77 per 1000 | 1 per 13 deaths |
| 1932 to 1934 | 191 | 63 6 per 1000 | 1 per 16 deaths |
| 1934 to 1935 | 174 | 58 per 1000 | 1 per 17 deaths |
| 1935 to 1937 | 168 | 56 per 1000 | 1 per 18 deaths |

## TREATMENT

### SPECIFIC THERAPY

*During* recent years many problems *in* the treatment of cardiovascular syphilis have been solved We can now formulate a scheme of therapy for each case upon much more firm ground than was formerly possible, when it was the prevailing custom to recommend the same program of treatment for all syphilitic patients The less specialized therapy of yesterday was attended by numerous accidents among the cardiac patients of the group who had early aortic involvement Vigorous treatment with arsenicals was

followed by sudden death in many cases while it produced in others a rapid onset of the symptoms of heart failure This led the medical pendulum to swing away from the use of the arsenicals in cardiac patients Today we are in the mid position and are again employing arsenicals in cases of uncomplicated syphilitic aortitis, but we have learned to be cautious and precede their use by adequate preparation with slower acting and safer drugs

The early accidents following the injection of arsenicals were due many times to therapeutic shock (Herxheimer reaction) caused by too sudden destruction of large numbers of spirochetes and the liberation of their endotoxins This was invariably attended by a sudden swelling or reaction in the aorta about the mouths of the coronary arteries Moore [74] believes that the Herxheimer reaction may cause a coronary occlusion a rupture of an aneurysm or if there is cerebral vascular involvement the sudden edema and infiltration may cause a cerebral hemorrhage In other cases the arsphenamine may prove too toxic for the already damaged heart muscle and cause ventricular tachycardia and fibrillation Vigorous approach with arsenicals in other cases may be followed by immediate clinical improvement but after this by a rapid downhill course (therapeutic paradox) caused by the scar tissue replacement in the luetic areas

A safe procedure to prevent these reactions is to begin treatment of the patient who has syphilitic aortitis with a heavy metal and iodide Bismuth is the heavy metal of choice First introduced in 1921 by Sazerac and Levaditi bismuth has succeeded in completely replacing mercury in the treatment of cardiovascular syphilis When given by intramuscular injection this heavy metal is slowly absorbed and excreted and a low but uniform concentration is maintained in the tissues As long as the bismuth is present the multiplication of the spirochetes is prevented although they are not destroyed This fact can be proved by animal experimentation While the exact mechanism of this action of bismuth on the spirochete is unknown the effect is not the same as that exerted by the arsenicals In the sense that it prevents multiplication of spirochetes in the syphilitic lesions thus allowing the defense mechanisms of the body aided by the iodides to heal the lesions bismuth may possess resistance building properties

The necessary concentration of bismuth can be maintained in the body by weekly intramuscular injections of one of the preparations of the insoluble salts The soluble salts are painful more toxic and require more frequent administration Consequently they are more expensive and less convenient for the patient I prefer bismuth salicylate a ten per cent suspension in oil The dose is 0 2 Gm (2 cc ) once weekly This preparation causes very little local discomfort and toxic reactions following its use are rare

Intramuscular injections of bismuth preparations are best given into the inner angle of the upper outer quadrant of the buttock (Fig 92) using a 2 to 2½ inch (21 gauge) needle on a 2-cc syringe The drug is

introduced deep into the body of the muscle Since an insoluble preparation is being used an aspiration test is essential before the injection is made to detect a chance entry into a deep blood vessel Following the *injection the site should be massaged for a minute to favor distribution*

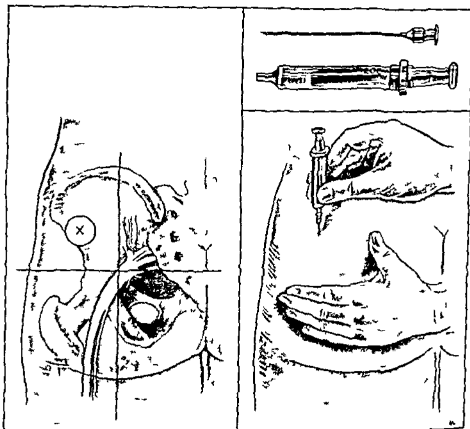


FIG. 9 Equipment and technic for intramuscular injections
A cc syringe (21 gauge) ½ inch needle
B Site of injection inner angle of upper outer quadrant
C Technic of injection

and better absorption of the drug Sterile abscesses following bismuth salicylate in oil are infrequent

Early in the course of bismuth therapy *a bluish gray line may appear* at the gum margins Less often a stomatitis appears, particularly in patients whose oral hygiene is poor Few alterations in renal function have been observed following a course of bismuth in the usual dosage and clinical evidences of liver damage are rare Aching of the bones and muscles quite like that produced by grippy or influenzal infections is *often complained* of during bismuth therapy, but its cause so far is unknown *Seldom do*

Herxheimer reactions occur when bismuth is used in the treatment of cardiovascular syphilis On the whole the toxic effects produced by bismuth compared to those observed following mercury and arsenicals are few

Intramuscular injections of bismuth iodide therapy and later the cautious use of intravenous injections of neoarsphenamine comprise the usual procedures of treatment in cardiovascular syphilis Lately a preparation of bismuth for oral administration has appeared on the market This semisolid termed sobisminol mass, is a complex organic bismuth product the exact chemical nature of which has not been fully established [33] It is obtained by the interaction of sodium bismuthate tri isopropanolamine and propylene glycol and contains between 19 25 and 20 25 per cent of bismuth The recommended adult dose consists of two to three capsules each representing 150 mg of metallic bismuth taken with plenty of water at 10 A M 3 P M and 8 P M When continued in this dosage for from 10 to 12 weeks they represent a course of bismuth therapy For children the dose is one capsule three times a day Such oral administration has been shown to produce a blood concentration of bismuth comparable to that following intramuscular injections Satisfactory antisyphilitic concentrations of bismuth are also found in the urine and cerebrospinal fluid [67]

Sobisminol mass given orally may produce gastro-intestinal upsets in some patients occasionally severe enough to cause them to discontinue the drug This is a disadvantage Bismuth stomatitis may appear and a bismuth line has been noted in some instances When the treatment is placed in the hands of the patient dosage is less accurate and there remains the tendency on the part of the patient to neglect to take the medicine as soon as improvement occurs Furthermore the physician is not apt to have as frequent contact with his patient and this gives less opportunity to observe either the untoward effect of the drug or the progress of the cardiac lesion No sustained bismuth effect is derived from the use of sobisminol by mouth since the excretion of bismuth in the urine falls off rapidly as soon as the capsules are discontinued Relapses therefore occur more readily since no bismuth depot is established when the oral route is used When intramuscular injections are used the full amount of the drug is administered

Occasionally some patients for business or professional reasons find it impossible to report to the physician at weekly intervals Likewise in rare instances intramuscular injections have to be discontinued because of pain and induration in the muscles *In these cases the temporary oral administration of bismuth is useful*

If oral bismuth therapy gains headway in the future the distribution of preparations of the drug should be carefully controlled Oral administration *should* remain under the physician s supervision since self medication in cardiovascular syphilis is extremely dangerous

The use of iodides in the treatment of cardiovascular syphilis is based upon the local healing effect of these drugs If given alone at any stage of the disease they have little influence on the spirochete When combined

with a heavy metal or an arsenical symptoms are more speedily controlled and the local healing produced by the iodide allows the more powerful antisyphilitic drugs to penetrate deeply into the tissues A similar effect on necrotic tissue is seen when iodides are used in other granulomatous diseases especially actinomycosis

When administered by mouth the absorption of the iodides is rapid Either the potassium or the sodium salt may be used although the latter is more expensive It is best to prescribe iodides in concentrated solution

| | |
|---|---|
| KI | 50.0 Gm |
| Water | 50.0 cc |

In this prescription one drop will contain approximately 0.065 Gm (1 grain) of KI The required dose should be dropped into a full glass of water or milk and given just before meals The usual dose of the above prescription is 2 to 4 Gm (30 to 60 grains) three times a day Iodides should be administered for periods of three months twice a year in combination with the heavy metal and not with neoarsphenamine When tuberculosis or simple goiter complicate the picture the administration of iodides is contraindicated

Some patients show an intolerance to iodides Coryza and an unpleasant brassy taste in the mouth may be disregarded However if gastro-intestinal irritability develops or severe acneiform eruptions appear, the drug should be at least temporarily discontinued In some instances the healing action of the iodides on syphilitic tissue with subsequent absorption of necrotic products may produce fever which subsides when the drug is withdrawn

After the preliminary course of 12 weeks of bismuth and iodide neoarsphenamine may be started cautiously Doses of 0.1 Gm (intravenously) should be administered at first and the amount gradually increased until 0.45 Gm is given at the fifth dose of a ten injection series [67-91] Arsphenamine should never be used in cardiovascular syphilis

Many variations are possible in the therapeutic schedule in this type of syphilitic involvement It is difficult to recommend a schedule that will fit every case Experience will soon show that each individual presents certain rules of his own and the most successful result is achieved by the physician who learns to follow them The age and build of the patient the duration of the infection the amount of previous treatment and the social status are all factors of importance that must be considered In short, in order to get the best result the physician must be skilled in the art of medicine and be able to interpret the symptoms of the patient in terms of the structural changes He should also be quick to detect any reactions that call for a pause in therapeutic activity

If congestive failure is present when the patient is first seen the usual measures described in Chapter 2 are carried out Digitalis should be given to the point of digitalization and then continued in a maintenance amount The organic mercurial diuretics are useful Many of these patients

are unfit for heavy metals when first seen but if edema clears and balance is restored bismuth and potassium iodide may be started

If aortic regurgitation is present and congestive manifestations have not appeared after the 12 weeks of preparatory treatment using bismuth and iodide neoarsphenamine may be cautiously started using an initial dose of 0 1 Gm and increasing this dose gradually depending on the body weight until a maximum of 0 45 Gm is reached in 12 treatments at weekly intervals It is most important to avoid reactions of all types with this course of treatment If reactions occur arsenicals should be discontinued and after a short rest period intramuscular injections of bismuth should be given If no reactions are encountered and the course of arsenicals is completed heavy metal and iodide for 12 weekly doses are again indicated In the absence of untoward symptoms these courses may be given alternately over a period of two years In each case the cardiac lesion is the guide to treatment and frequent examinations of the heart should be made

When angina is present and syphilitic involvement of the coronary arteries is suspected the prognosis is grave It is well to send these patients to the hospital if possible Bed rest and potassium iodide supplemented by the usual measures directed toward the relief of the angina are called for (Chapter 7) If improvement occurs heavy metal may be added I have never used arsenicals in this group of cases

In the presence of aneurysms great care should be taken to guard against reactions Heavy metals preferably bismuth should initiate the treatment and should be continued with iodides for ten weeks If arsenical therapy is used start with exceptionally small doses (0 025 or 0 05 Gm ) and increase slowly to 0 2 Gm in a 12 dose series These patients should be examined frequently and carefully (see Case 98)

As stated in the recent summary of the Co operative Clinic Group[68, 69] the best treatment is prophylaxis At least 30 injections of an arsenical and 60 injections of interim heavy metal (bismuth) administered under the continuous system while the patient is in the early stages of syphilis is after all the best form of therapy for the prevention of cardiovascular involvement This has been shown by Thompson and his co workers in a survey of 260 individuals who contracted syphilis 15 to 25 years before the study was made The incidence of cardiovascular involvement in the group was found to be 10 per cent a figure that agrees with the observations of other investigators However in the group studied by Thompson Comeau and White[374] all of the cases showing evidence of cardiovascular syphilis gave a history of inadequate early treatment

With treatment carefully planned and carried out a marked symptomatic relief is possible in patients suffering from syphilitic invasion of the cardiovascular system Proper therapy increases the average duration of life in the presence of uncomplicated syphilitic aortitis from 34 to 85 months[68] The average duration of life in patients who are treated with

small doses of arsenicals exceeds the duration of life of patients who are treated with large doses

### Treatment of Aneurysms

#### Wiring Carotid-Jugular Anastomosis

(See page 229)

## ILLUSTRATIVE CASES

### Untreated Syphilitic Cardiovascular Disease—Aortitis, Aortic Regurgitation and Cardiac Enlargement—Death Following First Attack of Congestive Failure—Autopsy

*Case 29* J M a colored laborer of 43 was admitted to the Philadelphia General Hospital /7/31 complaining of shortness of breath swelling of the legs and pain in the chest

History The dyspnea appeared a year before admission and gradually progressed to orthopnea A month before admission precordial pain was noticed It was referred to the left shoulder and appeared on slight exertion Edema of the feet in the evening came on six weeks before admission this increased rapidly and was generalized when the patient entered the hospital

Past History Chancre at the age of 18 No treatment

Physical Examination Cyanosis orthopnea anasarca B P 170/0 pulse 130 regular and Corrigan in type The heart was enlarged to the left (L B 16 0 cm) There was a diastolic murmur over the aortic area and absence of breath sounds accompanied by a flat percussion note over the right lower chest below the scapular angle (Fig 93A)

Laboratory Data Blood count normal Wassermann reaction positive The urine showed a cloud of albumin with hyaline casts

The roentgenogram showed generalized cardiac enlargement a wide aorta and fluid at the right base

The electrocardiogram showed a left axis deviation inverted T1 low voltage QRS and slightly prolonged P R intervals

Clinical Diagnosis A Etiologic Syphilis B Anatomic Cardiac enlargement Aortic regurgitation Aortitis involving the coronary ostia C Physiologic Normal sinus rhythm Anginal syndrome Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

Course The patient died suddenly on the fourteenth hospital day

Autopsy (Fig 93B) There was considerable cardiac hypertrophy The aortic valve leaflets were bound down to the aorta by syphilitic scar tissue The thoracic aorta was dilated and the seat of striations typically luetic The coronary orifices were stenosed but the coronary arteries themselves were normal throughout their course

Discussion On admission this patient presented all the signs and symptoms of advanced congestive failure The search for the etiologic background was a short one An initial lesion at the age of 18 with no subsequent treatment the negative rheumatic history the race, the age the evidence pointing to aortic regurgitation as the single lesion and the positive Wassermann reaction made the diagnosis of syphilitic cardiovascular disease inescapable

The usual rapid progress in the heart failure is evident Symptoms first appeared six weeks before death The anginal pain complained of on slight exertion suggests involvement of the coronary ostia in the syphilitic process

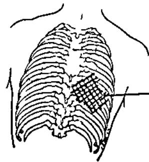






FIG 93 Syphilitic card ovascular disease Syph l tic aortitis Note aort c d latat on and marked card ac hypertrophy (Autopsy No 839 Philad lph a General Ho pital )

consequently the blood supply to the myocardium must be limited This may have been an important factor in the rapid downhill course and may explain the failure of the usual therapeutic measures to restore compensation

The history of the untreated primary lesion at the age of 18, and the long period of apparent latency of the infection (25 years) followed by rapidly developing signs of cardiac failure at the age of 43 are not uncommon Syphilitic involvement of the heart and aorta can exist in the absence of symptoms for many years as this man's story illustrates The development of aortic regurgitation paved the way for the cardiac failure and the coronary involvement hastened the end The patient, during the development of the disease, had no occasion to seek medical advice He felt well and continued to work as a laborer When symptoms finally developed at the age of 42, they succeeded one another rapidly

When cardiac failure appeared and the patient was admitted to the hospital the heart and not the syphilis demanded attention The measures employed in these cases are the same as in any case of congestive failure (Chapter 2) Angina should also be treated in the usual manner (Chapter 7) If severe anginal pain is present and is unrelieved by ordinary medical measures alcoholic injections are to be considered (page 256)

### Rheumatic Heart Disease Mistaken for Syphilitic Aortitis in Presence of Positive Wassermann Reaction—Autopsy

**Case 30** C B an Italian shoemaker of 50 entered the Philadelphia General Hospital on 5/13/34 complaining of chest pain and increasing shortness of breath

History Dyspnea was present for some years but became worse the month before admission and was soon followed by edema Chest pain on exertion was noted three weeks before admission

Past History Negative for syphilis Positive for rheumatism at age 5

Physical Examination B P 180/30 Orthopnea Edema of legs Rales at both lung bases Apex of the heart in the sixth interspace 15 cm to the left of the midsternal line Rhythm irregular Rate 100 Heart sounds were of poor quality Harsh systolic and diastolic murmurs were heard over the aortic area There was a long low pitched diastolic murmur over the mitral area

*Laboratory Data* *Blood Wassermann positive Urine showed* a cloud of albumin with many hyaline casts

The electrocardiogram showed many ventricular premature beats and a left axis auricular fibrillation

The patient d ed on the fifth hospital day

Clinical Diagnosis A Etiologic Syphilis B Anatomic Cardiac enlargement Aortic regurgitation C Physiologic Numerous premature ventricular contractions D Functional Classification Class 4 Therapeutic Classification Class E

Autopsy Both ventricles were dilated and hypertrophied The aortic and mitral valves showed the presence of an old rheumatic endocarditis The aorta showed very few healed syphilitic scars

**Discussion** In addition to the signs of congestive cardiac failure in this patient there was evidence of heart disease of long standing cardiac enlargement and the murmurs over the apex and the aortic area A Corrigan

type of pulse was present and the patient was found to have a positive Wassermann reaction Over the region of the cardiac apex there was a low pitched rumbling diastolic murmur This did not excite suspicion and was considered to be an Austin Flint murmur Consequently the etiologic background was thought to be syphilis Response to treatment was poor which seemed to confirm the diagnosis and the patient died on the fifth hospital day

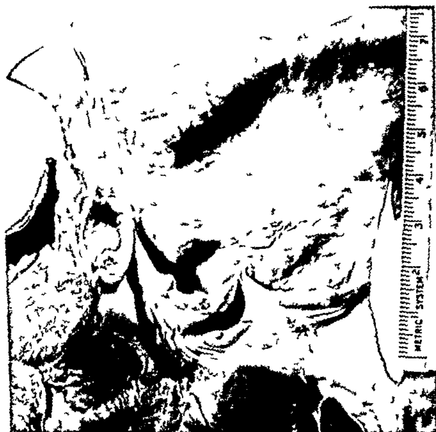



FIG 94 Rheumatic involvement of the aortic valve He led syphilitic aortitis (Autopsy No 7 596 Philadelphia General Hospital)

The main lesion found in this man s heart at autopsy was rheumatic (Fig 94) although the pathologist found some slight evidence of syphilitic aortitis

Reviewing this case in the light of the autopsy findings we must realize that at times it may be impossible to ascertain the exact etiology of a cardiac lesion After all the clues offered by clinical and laboratory studies

*are fully investigated, considerable doubt may still exist* Aortic valve lesions when they occur alone cause most confusion (page 204) However, the systolic and diastolic murmurs over the aortic area the typical rumbling diastolic murmur at the apex the positive rheumatic history and the duration of the dyspnea should make us consider rheumatic heart disease The positive Wassermann the aortic lesion and the sudden death of the patient during the first episode of congestive failure suggested syphilis as the background before the autopsy

Calcific aortic stenosis should also have been considered in this case Evidence of calcification of the aortic valve on the roentgenogram (see *Fig 26*) *would have strengthened a rheumatic diagnosis* The presence of the systolic murmur over the aortic area if accompanied by a thrill is usually evidence in favor of rheumatic etiology although *occasionally aneurysmal dilatations may produce a thrill in this area*

In this patient evidence of syphilis in other parts of the body wa entirely lacking in fact the Wassermann reaction alone seemed to sway *clinical opinion toward syphilis*

The electrocardiogram was returned to us after the autopsy In keeping with our impression of syphilitic heart disease we believed the irregular rhythm was due to frequently recurring premature beats However *the tracing showed the presence of auricular fibrillation an arrhythmia* much more likely to complicate the course of rheumatic heart disease than luetic

Occasionally cases in which an etiologic diagnosis of the aortic lesion cannot be made develop fever and other evidences of subacute bacterial endocarditis However while pointing to a rheumatic background in most cases, this complication is possible in congenital luetic or rheumatic types in fact it should always be kept in mind when discussing the cause of death of any cardiac patient Maher and Plece [5] have reported 12 instances from a series of 5000 cases in which syphilitic heart disease was complicated by the appearance of thyrotoxicosis This added burden increases the incidence of auricular fibrillation and congestive failure Successful surgical treatment of the thyrotoxicosis is usually followed by considerable clinical improvement *The mortality following subtotal thyroidectomy was sur prisingly low* in the series observed by Maher and Plece

A final point of importance in arriving at the correct etiologic diagnosis *so essential before proper treatment can be begun is the possibility of* rheumatic and syphilitic infections co existing in the same heart Cases of this type illustrated by the patient now under discussion are by no means rare

The treatment of this patient was first directed toward the cardiac failure If this had cleared the importance of establishing the correct etiologic background for the heart lesion at once becomes evident The treatment of the syphilis *would then have proceeded cautiously* with the cardiovascular system *in mind followed by frequent examinations* to ascertain the effect on the cardiac lesion

## Syphilitic Aortic Aneurysm of Transverse Arch (Aneurysm of Symptoms)—Steady Down Hill Course with Cough and Dysphagia—Autopsy

**Case 31** R A., an unemployed colored male of 53 was admitted to the Philadelphia General Hospital on 5/ 0/ 9 complaining of pain in the chest and shortness of breath



Fig 95 Large aneurysmal sac arising from ascending aortic arch Note the small size of the heart compared to that of the aneurysm (Autopsy No 0 8 0 Philadelphia General Hospital)

History Chronic cough for six years Increasing hoarseness and dyspnea followed by dysphagia and a loss of weight of 40 pounds appeared during the y a before admission

PHYSICAL EXAMINATION Marked dyspnea B P 110/80 on the right and 0/0 on the left Limited expansion of the left side of the chest Fullness over the left upper chest The breath sounds were distant over this area and exaggerated over the right chest There were rales at both lung bases There was a systolic murmur over the cardiac apex No cardiac enlargement A2 greater than P2

LABORATORY DATA Wassermann positive The roentgenogram showed a large aortic aneurysm The trachea was displaced to the right

Electrocardiogram normal

Urine showed a trace of albumin

CLINICAL DIAGNOSIS A Etiologic Syphilis B Anatomic Large aortic aneurysm C Physiologic Normal sinus rhythm D Functional Classification Class 4

The patient died suddenly on the 12th hospital day

AUTOPSY The heart was small compared to the size of the aneurysmal sac (Fig 95) About one inch above the aortic valve the aorta was seen to expand into a large aneurysmal sac measuring 15 × 16 × 10 cm and involving the remainder of the ascending and all of the transverse arches The sac was filled with thrombus and was attached to the apex of the left lung which was pushed forward and compressed There was likewise compression and erosion of the esophagus with a large ulcer The pulmonary artery where it divides and passes into the lungs showed constriction resulting from the aneurysm

**Discussion** The complaints of dyspnea cough and dysphagia focused attention at once on the mediastinum in this case and suggested aneurysm The clinical examination and the roentgen study quickly confirmed the diagnosis

Patients with aneurysm in this location lose considerable weight (aneurysmal cachexia) This may be a result of the continued pain which causes a loss of sleep In the case under discussion the dysphagia appeared to be an additional and perhaps a more direct cause of the weight loss Compression of the lung and trachea with atelectasis contributed to the dyspnea at the same time producing cough and hoarseness

The autopsy showed nothing to explain the patient s sudden death However some notice must be taken of the fact that the pulmonary artery was markedly compressed by the aneurysmal sac Encroachment in this area if long continued can in itself lead to cardiac failure through the production of cor pulmonale (page 426) Had this patient survived for a longer period clinical evidence of this pressure in the lesser circulation would have appeared in the form of cyanosis engorged pulsating jugulars, increasing dyspnea enlarged liver and the signs that we interpret as evidence of right sided heart failure Occasionally the dilated aorta may rupture into the pulmonary artery with the production of an arteriovenous aneurysm A loud prolonged murmur accompanied by a thrill then appears over the pulmonary area dyspnea increases and hemoptysis may occur This accident does not cause immediate death in fact, as we have seen in Case 6 the patient may live long enough to develop secondary changes in the wall of the pulmonary artery

The patient under discussion received a liquid diet and sedatives in sufficient quantities to relieve the cough and dyspnea The heart was not enlarged and aside from the dyspnea and slight cyanosis which could not

be considered entirely cardiac in origin there were no signs of cardiac failure Digitalis was not prescribed

Bismuth salicylate injections (page 214) and iodides by mouth were given for the effect they might have in reducing the pain by their specific action but were unsuccessful

SYPHILITIC AORTITIS AND ANEURYSM—EXCELLENT RESPONSE TO THERAPY*

**Case 32** E H a well nourished white woman of 51 consulted her physician in January 19 9 because of severe pain in the left shoulder chest and upper arm Her husband d ed a month previously of a ruptured aortic aneurysm

PHYSICAL EXAMINATION B P 14 /90 There was an increase in the area of supra cardiac dulness noted and a marked accentuation of the aortic second sound

PAST HISTORY Her blood Wassermann was positive in 1919 at hich t me appro priate therapy as begun but discontinued because of a reaction the exact nature of which was not clear No treatment was received between 1919 and 19 9

The roentgen examination (1929) (Fig 96A) showed d latation and increase in expansile pulsation of the first portion of the arch of the aorta The greatest width of the aortic shadow was 8 5 cm

CLINICAL DIAGNOSIS A Etiologi Syph l s B Anatomic Ao titis Early aneurysm of the ascend ng arch C Physiologic Normal sinus rhythm D Funct onal Classificat on Class

SUBSEQUENT COURSE Weekly inject ons of 50 m lligrams of bismuth salicylate in oil were given intramuscularly and increased unt l the patient was receiving 100 milligrams weekly

By the end of the fifth week of this treatment the chest pain had entirely disappeared The full course was completed

The roentgen exam nation w s repeated in three months and showed a definite decrease in the size of the aorta (Fig 96B)

At the end of the first year of bismuth therapy the patient felt so well that she di continued treatment again against the advice of her physic an

In August 1932 two years later she reappeared complaining of slight pa n in the left chest An orthodiagram at this time showed l ttle change The electrocardiogram (F g 96C) showed only a left axis dev ation

B smuth therapy was again started and a full course g ven w th appropr te rest periods over the next thre years

Follow up exam nation in 1940 showed little change The patient remained free of symptoms She had lived a normal life in every espect s nce the d agnosis was made in 19 9

DISCUSSION Chest pain in a patient of this age who has a positive Wassermann reaction and roentgenographic evidence of aortic involvement disqualifies her for neoarsphenamine unless preceded by a long preparatory period Many younger patients might show rapid improvement in their lesion following administration of neoarsphenamine but the physician might later regret this therapeutic triumph in the presence of a failing heart

The relief of pain in older patients showing this degree of aortic involvement is usually accomplished by persistent antisyphilitic treatment combined with the proper amount of rest Where larger aneurysms are present and are accessible the procedure of wiring and electrolysis (page 229) proves helpful in relieving the pain The various procedures recommended

* Courtesy of D Carroll S Wright

to block the nerves by *paravertebral injection* (page 256) are occasionally useful in the treatment of severe pain that attends aneurysmal growth



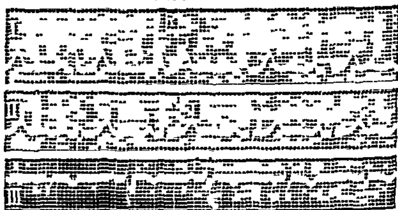




FIG 96 A Roentgen examination at beginning of treatment (19 9) Note increase in size first portion of aortic arch B Three months later Not decrease in size of the aorta

C The electrocardiogram (193?) showed a left axis deviation The other features like ise normal for patient of this age Note the change in voltage of QRS3 caused by respiratory influence

The relief of pain when specific treatment was begun in this patient was evidence in favor of the diagnosis of syphilis Iodides in large doses are useful in early aneurysm An initial dose of 0 3 to 0 6 Gm (5 to 10 grains) three times daily was prescribed in this case and gradually raised to 4 0 Gm (60 grains) three times daily

The absence of cardiac enlargement and aortic insufficiency in this case when treatment was begun contributed in no small measure to the good result The aneurysm was recognized early and the patient aside from occasional lapses in treatment was able to follow an ideal regime She never had to work for her living consequently occupational stress and strain which are factors of importance in the production of large aneurysms in the male were entirely avoided It is doubtful if the same treatment in the case of an aortic lesion of similar extent and duration in a negro laborer would have the same happy result This is one reason I believe why various statistical studies differ with regard to efficacy of treatment at this stage of the disease

### Cardiovascular Syphilis—Aneurysm of Ascending Aorta—Survival for Three Years After Wiring

**Case 33** J G a negro laborer of 56 was admitted to the Philadelphia General Hospital complaining of shortness of breath and chest pain

History Marked dyspnea and pitting edema were present on admission Dysphagia and chest pain of intermittent character present for six months

Physical Examination B P 150 60 Obese deaf colored male Signs of old hemiplegia There was a large pulsating prominence the size of a lemon to the right of the sternum extending from the second rib to the fourth intercostal space (Fig 97A) There were systolic and diastolic murmurs over the base of the heart A tracheal tug was present The liver was 4 cm below the costal margin

Laboratory Data Wassermann reaction negative

Electrocardiogram left axis deviation Occasional premature beat of ventricular origin

Roentgenogram large aortic aneurysm

Course The aneurysm was wired twice with 15 feet of gold wire at intervals of two years The patient died suddenly from rupture of the aneurysm through the anterior chest wall

Autopsy (Fig 97B) The heart was enlarged with left ventricular hypertrophy The ascending aorta and the arch were dilated There was a large aneurysmal sac just anterior to the origin of the innominate artery which had eroded the second third and fourth costal cartilages and the right border of the sternum The sac was filled with organized thrombus and firm clot Coils of wire were recovered from the center In the descending aorta a second aneurysm was found just beginning to erode the body of the fourth thoracic vertebra The remainder of the aorta showed both luetic and atheromatous changes

#### TREATMENT OF ANEURYSMS OF THE THORACIC AORTA

**Discussion** (Dr Henry D Jump*) The great majority of aortic aneurysms are due to syphilis The percentage will be found to vary in accordance with the observer and the consideration given to the history of infection the character of previous treatment the accuracy of the blood and spinal fluid examinations and the pathologic findings It may be accepted as a general rule that the farther from the heart aneurysms occur the lower will be the percentage that are caused by syphilis

From the nature of things the treatment of aneurysms is not very satisfactory and the most that we may expect to accomplish is palliation of

* Emeritus Professor of Applied Therapeutics Woman's Medical College of Pennsylvania




Fig 9 A Large aneurysmal sac presenting on anterior chest wall
B Large aortic aneurysm Note aortic dilatation and the presence of both atheromatous and syphilitic changes (Autopsy No 17 557 Philadelphia General Hospital)

symptoms and a slight prolongation of life The vessel is usually badly diseased and another part may be expected to break down at any time Often multiple aneurysms are present

In the postmortem room saccular aneurysms are usually found partly filled with laminated clot The most of these have small orifices which cause slowing of blood current and favor coagulation Blackmore and King[30] found six in a series of 42 postmortem specimens of saccular aneurysms to be filled completely with clot of 19 fusiform aneurysms seven showed concentric clotting which had narrowed their lumen to a diameter equal to or less than that of the aorta With such evidence at hand of Nature s way of spontaneously eliminating these dangerous vascular dilatations we are prompted to devise methods that may be expected to aid her and if possible speed the process

The following measures have been advanced from time to time the introduction of needles into the aneurysmal sacs the application of a galvanic current to the outside of the aneurysms the placing around the vessel of constricting bands to slow the blood current a dry diet with prolonged rest the use of antisyphilitic drugs the introduction of wire and other substances and the anastomosis of the carotid artery and internal jugular vein Only the last four have survived and are occasionally useful in the modern treatment of properly selected cases

Inasmuch as early treatment produces far better results diagnosis of aneurysms in their incipiency is much to be desired This is not easy for often a positive history of syphilitic infection is not obtainable there are no positive serum reactions and there may be no symptoms On the other hand routine physical examination may reveal a suspicious area of supra cardiac dulness which the roentgen examination shows to be caused by a dilatation in the region of the aorta Under the fluoroscope this mass may be seen to pulsate or if the aneurysm contains much clot or is firmly tied down by an associated periaortitis or mediastinitis of syphilitic nature pulsations may be absent The administration of specific remedies may ultimately cause resolution of these tissues and permit pulsations to be observed

As the lesion develops other signs and symptoms occur bruit pressure and pain diastolic shock pulsation in the interspaces enlargement of superficial vessels of the chest hoarseness cough tracheal tug pulsus differens etc

*The abdominal* aortic aneurysm is always hard to detect even when large It usually lies high in the abdomen and percussion dulness may not be elicited in the back because of the overlying liver In this region pulsation is very deceptive for often a pulsating aorta in a thin individual may simulate the expansile pulsation of an aneurysm However if the hands can grasp the upper and lower poles of the sac an expansile pulsation will be detected and this common error avoided A roentgen study may likewise be of considerable help Erosion of the vertebrae may be present and if the

mural clot has become calcified this will outline the periphery quite clearly Under the fluoroscope *expansile pulsations may be seen*

Diet and Rest This regime of treatment was offered by Valsalva Bellingham and Tufnell of Dublin in turn The name of the last investigator is still attached to the method Complete physical and mental rest were planned in association with the very low diet The object was to concentrate the blood slow the heart rate, and reduce the arterial pressure in the hope that coagulation within the sac might be favored (Chapter 21)

The Tufnell diet is made up as follows
*Breakfast* two ounces of milk, two ounces of bread with a little butter
*Dinner* three ounces of meat without salt and four ounces of milk For a portion of the milk one or two ounces of claret may be substituted
*Supper* the same as breakfast

This severe program few patients can be induced to accept today, since the anemia and weakness which follow its prolonged use add little to the patient s comfort The addition of considerable protein and a little more liquids will make it more acceptable, and should not interfere with its purpose After all it is not unlikely that under this regime there is an increase in the mural clot which reinforces the wall of the sac Iodide of potassium was also given with this program to 'promote coagulation'

*Antisyphilitic Drugs* The treatment of syphilitic cardiovascular disease in general and the modifications called for in aneurysm have been discussed (page 219) A few important points, however will be repeated here for emphasis

Reactions to treatment should be avoided These therapeutic shock' (Jarisch Herxheimer reaction) and therapeutic paradox' (Wile) occur most frequently after the use of arsenicals Reactions may usually be avoided by beginning treatment with iodide and bismuth and continuing for 10 to 12 weeks Arsphenamine should never be used Neoarsphenamine has a place in the treatment of cardiovascular syphilis only after preliminary treatment with iodide and bismuth The dose of neoarsphenamine should be initially 0 025 or 0 05 Gm with a gradual increase weekly to a maximum of 0 3 Gm

In about 50 per cent of aneurysms this treatment has been followed by *a relief of symptoms and probably a prolongation of life* Arsenic affords a more certain relief from pain than iodide and the heavy metal This coincides with the observations of Moore et al and Padget and Moore [91] Moore and his co workers reported a mortality of 90 per cent among patients receiving little or no treatment and 40 per cent among those who had adequate treatment There was an average duration of life from the onset of symptoms of 19 months among the former and of 75 months among the latter The number of cases observed however was small Stokes [7] reports one case of aneurysm where roentgen ray examination showed only a dilatation of the aorta three years after continued treatment

In cardiovascular syphilis the serology is no guide to the efficacy of the treatment In only about 50 per cent of the cases is the reaction reversed

Heart failure occurs infrequently in aneurysm It may appear as a terminal event approximately 10 to 12 per cent die in this manner in contrast to 35 to 40 per cent who die of rupture of the aneurysmal sac When congestive failure occurs specific treatment should be discontinued and the cardiac symptoms treated in the usual manner with rest digitaliza tion and mercurial diuretics However the chances of improvement when congestive failure becomes established are small

WIRING OF AORTIC ANEURYSMS The introduction of wire and the passage through it of a galvanic current for the purpose of coagulating the blood offer a measure of relief in selected cases of sacculated aneurysm In 1864 Moore suggested the introduction of filiform material into an aneurysm to produce clotting and he accomplished it in one case This pioneer investi gator also stated that if wiring is to be done no artery should open from the aneurysm for if wire is exposed in a violent current of blood fibrinous clots will certainly break off from it and plug distant arteries He thus expressed the fundamental fact that only saccular aneurysms are sus ceptible to treatment by wiring

The next step was taken in 1879 by Corradi of Italy who used galvanic current through the wire he had introduced into a thoracic aneurysm There was marked relief for about three months The technic of Moore and Corradi has been the most acceptable and has been employed since its introduction Hare used it and reported a marked benefit in all of his cases although some had been in desperate condition previous to wiring Rosenstirn[3 3] reported a case in whom the improvement was remarkable A 25 year old patient had an aneurysm of the ascending arch which pro truded on the chest Severe pain required heavy doses of opiates while oxygen was given for frequent spells of suffocation A few days after wiring the pain began to improve in two weeks dyspnea was relieved and in six weeks the pulsating tumor subsided Later he was reported to be in most excellent health to undergo any ordinary exercise He lived for 11 years and eight months following the introduction of the wire Rarely do such remarkable results occur but in the cases I have wired improve ment in symptoms has always been observed

There were no changes in Hare s technic until Millar [?] experimenting with cats inserted various kinds of wire into the aorta and found that zinc caused a better coagulation of the blood than any other metal including gold and platinum He did not employ the galvanic current

Blackmore and King[30] devised an elaborate apparatus to introduce and coil a double strand of wire in the aneurysmal sac Their objects were to insert enough wire to slow the blood current and to heat this by an electric current to the point where the blood protein coagulates The coagulum that forms on the wire is difficult to scrape off This method was used in 11 cases with considerable success

INDICATIONS FOR WIRING Wiring is a procedure to be considered in saccular aneurysms only The object is to produce a clot in the aneurysm which will be attached to the wire and the wall and completely fill the sac If applied to fusiform aneurysms coagula which form on the coils of the wire may be swept off as emboli and lodge in distant vessels Moore pointed out this danger but in spite of his warning several fusiform aneurysms wired by the Moore Corradi method have been reported In each case emboli have formed and death has followed Blackmore and King have been able to use their method in the fusiform type of aneurysm with no untoward results

It was formerly stated that a thoracic aneurysm must protrude on the surface in order that a wire might be introduced with certainty into the sac However using a long needle guided by the fluoroscope it should be possible to reach those a slight distance below the surface In smaller aneurysms the results should be better for in these the aorta is less diseased than in the ones of larger size Even when blood oozes from the aneurysm that protrudes on the surface it may be successfully wired and external rupture delayed

The abdominal aneurysm must be exposed by celiotomy since few of them reach the surface I feel that the only indication for wiring the abdominal aneurysm is the great pain caused by their pressure on contiguous structures

CONTRAINDICATIONS TO WIRING When attacks of paroxysmal cardiac dyspnea are present the cases are almost hopeless and the operation of wiring will not postpone death Hare was of the opinion that the presence of more than one aneurysm was a contraindication The filling of one by a clot will deflect the blood current in such a manner as to increase the pressure in the other in which event rupture is *more apt to occur* For the same reason Hare believed that wiring should not be done in a saccular aneurysm which is distal to a fusiform

The equipment needed for wiring consists of

Solution of iodine 3 5 per cent
Alcohol
Hypodermic syringe and needles
*Hollow needle of about 0 gauge insulated with porcelain shellac or varnish except* 5 mm (1/4 inch) of the tip
Wire of gold alloy (gold 60 per cent silver 30 per cent platinum 10 per cent—gauge 14)
Galvanic instrument with rheostat and milliammeter which is connected with house current or to six or eight dry cells
Oval metal pad 0 x 30 cm (8 x 10 inches) covered with cotton pad
Small scalpel
Rubber gloves
Gauze dressings sponges applicators collodion

The procedure is carried out under strict antiseptic precautions The wire is wound upon a spool and sterilized by heat For an aneurysm 7 cm (3 inches) in diameter (on the roentgen plate) two to three meters (7 to 10 feet) will suffice For an aneurysm 10

to 13 cm (4 to 5 inches) in diameter three to four meters (10 to 15 feet) will be needed

The needle also is sterilized by heat if it is insulated with porcelain If covered with a heavy coat of varnish or shellac boiling will soften this but it will harden when cooled Soaking in pure grain alcohol will sterilize it but part of the insulation will go into solution

The operator and his assistant should wear rubber gloves and if the floor is not dry rubber soled shoes

After sterilizing the skin overlying the aneurysm with iodine and alcohol it is anesthetized with 1 per cent procaine solution and a small incision is made through the skin 1 to 2 cm away from the thinnest part of the aneurysmal wall The needle after being dipped into sterile water to lubricate it is quickly thrust through the incision into the aneurysm in a direction away from the opening of the sac *Blood will spurt intermittently from it immediately after the sac is entered Until this occurs the wire must not be introduced* If blood does not appear and the needle is not occluded a longer needle may be needed in order to penetrate the clot in the margin of the aneurysm Puncture at another point may be useful in reaching the interior of the sac Feed the gold wire slowly into the sac unwinding it carefully from the spool to prevent kinking The *positive* pole of the battery is next attached to the wire and the *negative* to the pad which has been previously moistened and put under the patient s back or buttocks or on the abdomen Turn on the galvanic current slowly beginning at 5 milliamperes and increase it 5 milliamperes every minute until 45 to 50 milliamperes are being given Continue this amount for 10 minutes and then gradually withdraw the current by reducing 5 milliamperes every minute Loosen the needle from the clot gradually and gently by turning and then slowly withdraw it using counter pressure on the sac Cut the wire close to the skin and push the end beneath it * Dress the puncture with collodion or sterile pad of gauze There is rarely any leakage Following wiring the patient should be kept in bed for a period of at least two weeks to encourage consolidation of the clot

The results that may follow wiring of an aneurysm are

1 Pain lessens or disappears soon after the current is turned off
2 Other pressure symptoms are relieved when the size of the aneurysm decreases
3 The size of the aneurysm decreases when the clot consolidates and becomes organized
4 Rupture of the aneurysm is delayed or prevented in many cases and the patient s life prolonged

The prognosis varies with each case and depends entirely on the condition of the aorta No deaths are known which can be attributed to the procedure of wiring

* It is important that the patient should not have access to the end of the gold wire This point was emphasized at a Philadelphia General Hospital autopsy During the period when the supply of the metal was limited Dr Jump sought to recover his wire from the inside of an aneurysmal sac When a careful search of the large firmly adherent clot as well as adjacent regions failed to show any trace of the strand that had been fed in so generously and truthfully the chemists were hurriedly called in consultation When the mystery deepened the physiologists were summoned However the wire had not been absorbed The bright youth from the Social Service Department returned the next day and upset all the hasty calculations His investigations revealed that the patient had found the end of the wire learned its composition and pulled it out inch by inch during frequent visits to a local tap room

W G L

In a few cases reported in literature, the wire has escaped from the sac and entered the aorta In one abdominal aneurysm on which I operated, the wire traveled upward until it reached the aortic orifice (Fig 98A and B) However, the patient's pain, which had been very severe was much relieved and the wandering strand of wire produced no untoward effects so far as we were able to determine The patient died 10 weeks later and we found at postmortem a rupture of the sac in the lower pole, which contained no clot The rest of the sac was well filled with a firm clot enmeshed in the wire There was no clot on the wire in the aorta and on close inspection no injury to the vessel was apparent

In recent years carotid jugular anastomosis has been the procedure of choice in the surgical treatment of aneurysm at the Philadelphia General

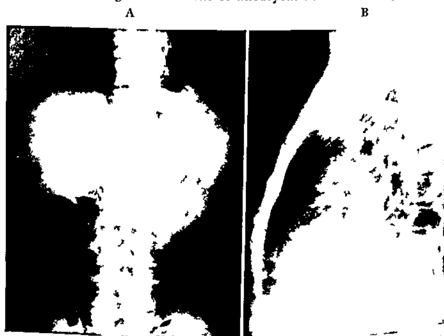



Fig 98 A Roentgen film showing coil of wire in aneurysmal sac B Same patient lateral view Note coil of wire in aorta as far as aortic valve

Hospital Since this procedure will be discussed in detail after the next case presentation my remarks concerning it will be few In 1930 McCarthy[4] reported ten cases and showed that the operation greatly relieves pain reduces the size of the aneurysm and postpones rupture In two of his cases where pressure symptoms recurred after primary relief I have resorted to wiring and in both, symptoms were again relieved In one patient a considerable reduction in the size of the aneurysm followed the wiring This major operation upon blood vessels that are the seat of advanced syphilitic disease in patients who are quite ill is less attractive to me than wiring The results are approximately the same following each procedure

## Cardiovascular Syphilis—Aneurysm of Aorta—Successful Carotid Jugular Anastomosis

**Case 34** Mrs M S age 59 was admitted to the Memorial Hospital on 1/5/39 complaining of smothering spells blood spitting pain in the chest and a persistent cough

History The patient was in good health until August 1938 when she had an attack of severe dyspnea followed by hemoptysis These symptoms subsided after rest in bed She had another attack in December 1938 About one week later she awoke at three o clock in the morning with a smothering feeling dyspnea and severe chest pain A hypodermic of morphine was required Following this seizure the patient was forced to remain in bed until she was admitted to the hospital Past history negative One child living and well No miscarriages

Physical examination revealed a well nourished elderly female propped up in bed coughing frequently and raising small quantities of blood streaked sputum

The left pupil was smaller than the right and did not react to light The carotid pulsation on the right side was quite noticeable No tracheal tug The chest was asymmetrical there was some bulging of the sternum at the second rib There was considerable widening of the area of supracardiac dulness

The heart was enlarged to the left The sounds were of poor quality the rhythm regular There was a systolic murmur at the cardiac apex another loud rough systolic murmur over the entire area of aortic dulness The blood pressure was 148/80

Laboratory Data Urinalysis revealed nothing abnormal Blood count R B C 4 040 000 W B C 6 650 hemoglobin 75 per cent (Sahli) N 50 M 7 F 1 B U N 9 mg per 100 cc of blood Glucose 100 mg The Wassermann reaction was four plus

Roentgen examination of the chest showed a heart enlarged in all diameters There was a large fusiform dilatation of the aortic arch mainly in its transverse and descending portions No erosion of the spine was evident in the lateral view

Clinical Diagnosis A Etiologic Syphilis Arteriosclerosis B Anatomic Cardiac enlargement Relative mitral insufficiency Syphilitic aortitis Aneurysm C Physiologic Normal sinus rhythm D Functional Classification Class 3 Therapeutic Classification Class E

Course Following a period of two weeks of bed rest during which time the patient was digitalized and given mercurial diuretics as required on 1/16/39 a left carotid jugular anastomosis was performed Her convalescence was uneventful Since operation there has been no hemoptysis dyspnea or pain

On 11/ 0/39 nearly a year later the patient reports no cough no pain and a marked increase in exercise tolerance B P 140/90 Bismuth and iodides were given during this period

Discussion (Dr James Lehman) In 1925 Babcock[18] described an operation for the relief of aneurysm of the arch of the aorta which has proved beneficial in many cases Heretofore all operations suggested or tried have been aimed at slowing or abolishing the flow of blood through the aneurysmal sac Carotid jugular anastomosis directs the arterial blood stream into the vein thus eliminating the peripheral bed of capillaries increasing the velocity of blood in the aneurysm and reducing the intra vascular tension

It is a well known principle of hydrodynamics that a liquid moving through a tube under pressure exerts pressure against the wall of the tube inversely as the velocity through the tube As the lumen of the tube is constricted the velocity increases and the lateral pressure decreases Con

versely as the lumen of the tube is increased the velocity decreases and the lateral pressure becomes greater It is the retardation of the flow of liquids through a tube that causes rupture not the high velocity Consequently when the aorta dilates to form an aneurysm the blood current is slowed and the wall of the aneurysm is subjected to much greater pressure than is the artery above or below this dilatation If it is possible to increase the velocity of the blood in the aorta and the aneurysm we should be able to decrease the lateral pressure on the wall of the sac sufficiently to prevent rupture




Fig 99 The steps in carotid jugular anastomosis

In 1930 McCarthy [1] reported ten cases operated upon by the Babcock method with gratifying results Since that time he has operated upon additional cases and states* that he has never observed rupture of an aneurysm following a carotid jugular anastomosis

The operation can be performed under local or light gas anesthesia The sternocleidomastoid muscle is divided transversely and the carotid sheath exposed Care should be taken not to injure the vagus nerve Umbilical tapes are placed above and below the point of anastomosis (Fig 99) The artery and vein are then divided and an end to-end anastomo is performed suturing intima to intima A second layer of reinforcing sutures should be placed to make the anastomosis secure The tapes are slowly removed and the wound closed in the usual manner

LARGE ANEURYSM OF TRANSVERSE AND DESCENDING AORTIC ARCHES—ONSET WITH BACHACHE AND PAIN IN LEFT ARM—ARTERIOVENOUS ANASTOMOSIS UNSUCCESSFUL—AUTOPSY

**Case 35** J G a colored laborer of 45 was admitted to the Philadelphia General Hospital complaining of pain in the left side of the chest and in the back of a years duration

* Personal communication



Fig 100

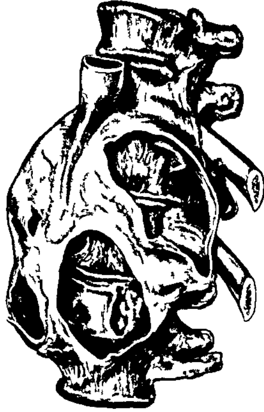


Fig 101

Fig 100 Large aneurysmal sac nvolving the transverse and the d sc nding portions of the aortic arch (Autopsy No 20 808 Philadelphia General Hospital )

Fig 101 Aneurysm of the d cend ng thorac c aorta Note th rosion f the vert brae with preservation of the ntervertebral d scs

History Patient unable to work for the past four months The pain started over the area of the left scapula and radiated to the left arm It was mad wor e by moving in bed and was relieved by lying on the right side Backache constant A loss of 20 pounds in six months was reported Untreated chancre 15 years before admis ion

Physical Examination Emaciated colored male of 45 B P 128/90 on the right and 60/0 on the left The radial pulse was weak on the left and full on the right side The pupils were small and fixed and did not respond to light The heart was not enlarged The sounds were of fair quality and a systolic thrill and systolic murmur were heard over the aortic area A was accentuated

Laboratory Data Electro ardiogram left axis deviation

Fluoroscopic examination dilatation of the aorta involving the transverse arch with slight dilatation of the ascending portion

*Clinical Diagno is* A *Etiologic Syphilis* B *Anatomic Aneurysm ascending and* transverse arches C Physiologic Normal sinus rhythm D Functional Classification Class 4 Therapeutic Classification Class E

Course Anastomosis of the left carotid and jugular vein was performed Fourteen hours later the patient developed a right sided hemiplegia and died

Autopsy The aneurysm involv d the entire transv r e and descending portions of the aortic arch (Fig 100) The bodies of the second third and fourth vertebrae were eroded (Fig 101) The int rvertebral discs were preserved The spinal cord was exposed The orifice of the left subclavian was covered by extension of a large clot in the sac The heart was normal

**Discussion** Lucke and Rea [39] in a study of 249 cases of aneurysm found erosion of the vertebrae in 53 and spinal cord compression in one In a series of 100 cases reported by Brindley and Schwab [4] only one aneurysm was found to involve the spinal cord although a number eroded the vertebrae to some extent Shimkin[338] has lately emphasized the fact that compression of the spinal cord is a rare complication of syphilitic aortic aneurysm In the patient whose history appears above in spite of the marked erosion of the vertebral bodies no signs of spinal cord com pression were elicited

*The duration of life in these cases does not usually* exceed two years from the date of the onset of symptoms although occasional instances of exceptionally long survival appear in literature Considering the severity of the lesion and the pain that invariably attends the erosion it is sur prising how long these patients continue to work before applying for treatment This patient at the onset of his difficulty took a great deal of medicine for neuritis Failing to gain relief he went to a chiropractor who gave him a series of treatments When these made him wor e he went to bed where he obtained the most relief He had been in bed for four months without medical care when he was removed to the hospital Morphine in large doses was then required to control the pain

At the end of the first week an arteriovenous anastomosis of the left carotid artery and jugular vein was done under local anesthesia Pain ceased following the operation but 14 hours later the patient developed a right sided hemiplegia and died

# 7

# ANGINA PECTORIS

In medicine there are instances where the most profound human wisdom is unable to anticipate the course or predict the outcome — CORVISART (1808)

The term angina pectoris was first used by William Heberden in 1768 in referring to the symptom of chest pain of the type that was later (1788) shown by Edward Jenner to be associated with disease of the coronary arteries It is important to realize at the start that angina pectoris as its name implies is merely a symptom and not a separate disease entity While many terms exist to confuse the picture (stenocardia precordial pain anginal pain retrosternal pain) they all refer to a paroxysmal sense of constriction or pain of short duration in the upper chest that is produced by any factor increasing the cardiac burden and relieved by nitroglycerine or rest When we use vague terms like anginoid pain or pseudo angina we are only hiding our ignorance for these create a feeling of false security that hinders proper treatment

Consequently after a careful study of the patient we should form an opinion as to whether or not the pain in question is cardiac in origin Many times this opinion will have to be based entirely on the patient's story for the rest of the examination may be negative The sensation in the chest is described in various ways by different groups of patients Some refer to it as a constriction others call it a rawness or burning Whatever sensation is complained of inquiry should be made as to its nature following exertion The patient soon learns that the quickest relief comes with rest and consequently remains motionless during subsequent attacks It is not necessary to elicit a history of radiation of the pain to the shoulder or arm before a diagnosis of angina can be made for often there is no radiation the sensation being confined to the upper sternum In other cases there may be a typical radiation to the arms usually the left the side of the neck the jaw face and in rare instances the back If the pain is intense it may be felt in both arms although the left arm alone is more frequently affected The pain usually descends on the ulnar side of the arm and hand (Fig 102) and is followed by a feeling of numbness in this same region Occasional reference to the abdomen may cause difficulty in diagnosis (page 478)

While angina is commonly induced by exertion it may follow excitement anger heavy meals sudden exposure to cold or overindulgence in tobacco or coffee The presence of one or more of these precipitating factors

may at any time be responsible for the increase in the number and severity of the seizures

Attacks of angina follow no set rule The first attack may prove fatal or the issue may be deferred many years The pain may last a few seconds to a few minutes and in some patients may not cause a great deal of restriction in ordinary activities Angina may vary from a slight ache or constriction of the chest to a pain of great intensity depending on the sensitivity of the nervous system of the sufferer Some individuals experi

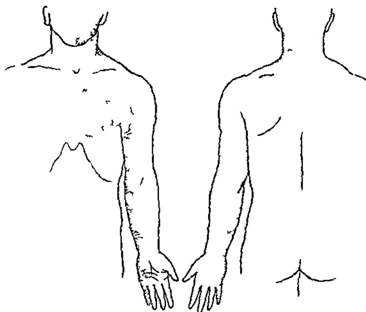


*FIG 10? The shaded areas in the diagram indicate the usual distribution of the* pain during an attack of angina pectoris (Redrawn from Diseases of the Heart Sir James Mackenzie London Oxford Univ Press *1918* )

ence severe pain on the slightest provocation while in others the symptoms may be mild less easily produced and readily controlled However the possibility of sudden death during an attack should be borne in mind in all patients once the diagnosis is established Some ambulatory patients report many slight attacks each day and consume large quantities of nitroglycerine in their relief, others have fewer seizures There may in fact be long intervals of freedom from the symptom but recurrence is the general rule In rare cases angina disappears entirely (page 276) It is also possible for coronary disease to progress to an advanced stage without the appearance of angina on any occasion, in fact the majority of patients with coronary artery disease do not have angina

When the nature of the chest pain is evident the prognosis occasionally may be estimated by careful study of the patient at subsequent visits If

the anginal pain comes on more frequently and is induced by less exertion we can infer that increase in the degree of coronary sclerosis has probably occurred in which event the outlook is more serious Eventually attacks may be induced by the slightest stimulus even when the patient is resting Unusually severe pain of the type indicating occlusion of a branch of the coronary tree may replace the anginal pain in these patients at any time

## INCIDENCE

**Sex** Angina is much more apt to occur in men than in women, a fact that is constantly observed in all statistical studies

**Heredity** as every practitioner knows is another very important consideration in the diagnosis of angina The sudden death of either parent or of a near relative from angina or coronary disease is a vote in favor of the diagnosis of angina when the nature of the patient s chest pain is in doubt Obesity hypertension and diabetes are common accompanying factors that appear in the same family groups too often to be merely coincidental The overweight hypersthenic man or woman in middle life who usually has a ruddy glow of health and an abundance of energy is the anginal type When chest pain is complained of by these individuals it should always be carefully investigated

**The age** of the patient is another clue in the diagnosis Angina is rare (particularly in women) under the age of 40 in the absence of diabetes or hypertension It is more common after 40 and its greatest incidence is between 50 and 60 Angina may occur in young people but is very rare in the absence of an aortic lesion (page 255)

**Race and Occupation** The symptom is common among Hebrews and rare among Negroes It is much more apt to occur among executives and skilled workers especially where the duties are accompanied by the nervous tension so characteristic of our present urban existence Angina is not as frequently met among races where quieter living is the rule particularly in the tropical climates

## ETIOLOGY

As the clouds roll away from the many controversies concerning the etiology of angina that have filled the 150 years since Heberden s time the prevailing belief seems to be that it is caused by myocardial anoxemia But the skies have not entirely cleared since the work of some recent investigators does not fully agree with this theory In any event anoxemia of the heart muscle can most plausibly account for the majority of our present-day views Angina may be compared to the symptom that we call intermittent claudication that arises from the calf muscles on exertion when their blood supply is reduced The most common cause of myocardial anoxemia is progressive narrowing of the coronary arteries by an arteriosclerotic lesion The same interference to the blood supply can be

caused by spasm in younger individuals but it is doubtful if this element plays an important part in older patients whose hearts at autopsy are seen to contain rigid calcified coronary arteries Increase in the aortic tension was advanced by Albutt to explain angina, but it has now been shown that increase in the blood pressure in these cases is by no means a constant finding Wenckebach weighs the possibility of increased cardiac action in the absence of peripheral dilatation as the main cause of the anginal attack and he cites the action of nitroglycerine on the peripheral vessels as proof of his theory

**Syphilis** is a factor in the production of angina only when the luetic lesion extends down far enough in the aorta to involve and constrict the coronary openings Consequently syphilis explains only a very small per centage of the cases

**Aortic incompetence** from rheumatic disease may decrease the cor onary blood flow and give rise to angina in young people (see Case 39) In severe anemia the blood entering the coronary circulation is much reduced in hemoglobin If the caliber of the vessels is still further en croached upon by arteriosclerosis symptoms may appear when any unusual exertion is attempted However with advanced degrees of anemia the patient s general physical condition is such that he is usually not inclined to participate in severe forms of exercise and consequently the level where pain appears is seldom reached and rarely exceeded

**Thyroid Overaction** The increased metabolism that occurs in patients with thyrotoxicosis places an extra load on the circulation including the coronary arteries If a mild sclerotic change is already present but is insufficient to cause a deficiency in the blood supply to the myocardium this extra demand imposed by thyroid overaction may be just sufficient to cause the appearance of angina

We can now see the variety of factors that may be responsible for the production of the symptom that we refer to as angina pectoris Conditions arising in the heart itself in other organs at a distance from the heart or alterations in the blood picture provided the nervous mechanism of the patient is sensitive to stimuli arising from the area of myocardial ischemia may all be the cause at one time or another of anginal pain Since the pathways of these pain impulses from the heart have become better known attempts at blocking them by alcohol injections or surgical removal of vital segments have been attended by increasing success (page 256)

## DIAGNOSIS

Since angina is a symptom arising from a purely functional disturbance we should not be surprised when all methods of clinical examination yield negative results Many patients suffering from coronary insufficiency will show no increase in the cardiac size a normal blood pressure and an unaltered electrocardiogram If the course is complicated by the occurrence

of one or more coronary occlusions characteristic signs are very apt to be found in the physical examination and the electrocardiogram If hyper tension is present there may be cardiac hypertrophy relative mitral insufficiency and the other manifestations described in Chapter 9 Occa sionally if the electrocardiogram is taken during an anginal attack tran sient alterations in the RS T intervals or T waves may be revealed that are characteristic of coronary disease (Chapter 24)

## PROGNOSIS

If syphilitic heart disease is complicated by angina we can say at the start that the prognosis is poor (page 220) The ease of production of pain may be a guide to prognosis in a patient showing little evidence of nervous instability As a general rule the easier the production of pain the poorer the prognosis When typical anginal attacks occur while the patient is at rest the outlook is not good In White s series[39, 39c] the average duration of life from the onset of angina to death was well over five years Macken zie s study [40] showed a duration of 5 4 years On the whole it can be said that the average duration of life from the onset of angina to death of the patient lies somewhere between five and ten years Exceptions occur and in rare instances the syndrome disappears entirely Many patients who suffer from angina eventually succumb to other complications of their arteriosclerosis in the brain (cerebral hemorrhage) kidney (nephrosclero-sis) or lung (pneumonia)

## TREATMENT

Successful therapy in angina depends a great deal upon the influence of the physician on his patient In the beginning care should be used in statements made to the patient about the condition The situation can be satisfactorily and completely explained without using the word angina Emotions are closely associated with heart disease and of all words in the language capable of exciting them angina probably leads the list

**Regimen of Life** The management of no phase of heart disease will tax the ability of the physician more than cases of angina since the suf ferer must first of all be made to accept life with a handicap In those who possess the proper philosophy to do this gracefully life expectancy may be considerably increased The visits to the physician are in large part made for the supervision of this period of readjustment in the habits of living The patient is studied to ascertain what activities can be carried out with safety and how these coincide with earning a living The proper amount of exercise should always be encouraged while the dangerous phases of the day s program must be omitted no matter what the cost Here there can be no compromise A skilled physician will have his way and remain the respected adviser of the patient while the latter continues

to maintain the interest in life that is so essential if progress in treatment is to be made The physician must encourage and inspire the patient at all times yet be able to restrain him from engaging in harmful activities without too much emphasis on the limitations The practitioner who tells the patient nothing and continually describes the incurability of the condition to members of the family is not long retained

It is most important to follow a definite regime of treatment in all cases and this must be outlined to suit the individual needs of the patient Something new if sufficiently conservative should always be injected into any plan when it becomes monotonous Above all the patient should never be allowed to drift along as a chronic incurable case If the physician has succeeded in kindling the hope in the future at the first interview he is very much at fault if he does not strive to carry out his side of the contract at all subsequent visits

A careful scrutiny of the patient s daily routine usually brings to light the activities that most often provoke attacks These should be forbidden in constructing a program that is planned to allow all the exercise that may be tolerated without pain The patient should be instructed to stop and rest when he feels an attack coming on and no task should be started where this cannot be done If an attack is experienced that tends to persist in spite of rest and medication the patient should be told to return home and take no further exercise until seen by his physician

It may be impossible to re educate some patients to live quiet and orderly lives to go about their work slowly and deliberately, to take time with their meals to secure the proper amount of rest at night and to arrange periodic vacations away from business Other cases require time patience and all the tact that the physician can muster

**A well balanced diet** should be prescribed It is also important to regulate the amount of food especially when obesity is present In some cases food at frequent intervals may reduce the danger of overeating at meal times and serve the additional purpose of maintaining the blood sugar level at a top normal figure Protein restriction is unnecessary in patients suffering from angina unless the condition is complicated by an advanced renal lesion

THE GLUCOSE AND INSULIN REGIME has been popular in some clinics in recent years but I do not use it in the absence of diabetes In diabetic patients where insulin is required care should be taken to avoid *hypoglycemic* reactions that are attended by an increase in the anginal pain (page 287)

Tobacco has no place in the treatment of angina and should be avoided entirely if the seizures are frequent and severe[399] Coffee and tea may be allowed in moderate quantities Warm climates are preferable for vacations when these can be arranged Moderately high altitudes usually produce no increase in symptoms in the average patient but some react badly (page 250)

## Drugs

Nitrites The therapy of the attacks themselves in addition to rest consists in the use of one of the nitrite group These drugs act by their vasodilating effect both on the coronary circulation increasing its flow and on the other arteries decreasing resistance to the blood flow and thus lessening the work of the heart The action is peripheral and not on the vasomotor center Members of this group of drugs that are in common use today are amyl nitrite sodium nitrite erythrol tetranitrate and nitroglycerine The last two are organic nitrates but are reduced to nitrites in the body and consequently have a characteristic nitrite action All the nitrite drugs above mentioned have a similar action but differ in the time it takes for this action to become manifest in the body

Amyl nitrite possesses the quickest action It is a volatile liquid obtainable in ampules (2 cc ) and is administered by breaking the ampule in a handkerchief and inhaling the drug The effect appears in a few seconds reaches its maximum in about two minutes and passes off entirely in about ten minutes Amyl nitrite is rapidly absorbed into the blood stream from the lungs and quickly excreted It relieves the anginal attack at once but is less convenient than tablets of nitroglycerine and has the added disadvantage of attracting attention to the sufferer in crowded places where attacks are most likely to occur

Nitroglycerine tablets (dose 1/100 to 1/200 grain) are usually preferable as they are more easily carried can be taken under the tongue unnoticed and are less expensive than amyl nitrite ampules Nitroglycerine is absorbed from the mucous membranes of the mouth and acts in less than two minutes if a fresh friable tablet is used The effect may last an hour It is always well to start the patient on a triturate containing a smaller dose (1/200 to 1/400 grain) for occasionally certain individuals possess an idiosyncrasy to this drug If untoward effects in the form of throbbing in the head faintness headache or even syncope occur it is difficult to persuade the patient to repeat the tablet in any dosage when the anginal pain recurs

Sodium nitrite (dose ½ to 1 grain) given in tablet form by mouth acts more slowly (15 minutes) and for this reason has no place in the treatment of an attack of angina However its action is more prolonged lasting in some instances for two hours hence the drug is useful in prophylactic therapy

Erythrol tetranitrate (dose ¼ to ½ grain) is an even slower acting drug than sodium nitrite but the duration of its effect is three to four hours

All nitrites produce a vasodilating effect that may be readily observed in the skin vessels particularly of the face where the skin temperature is elevated The pulse rate rises following nitrite therapy but this is purely a secondary effect following the fall in the blood pressure The nitrites have no direct cardiac action I never use any of the group for the purpose of constantly maintaining a lowered blood pressure level

**Trichlorethylene** has been recently used in preventing attacks of angina in doses of 1 cc by inhalation [37] The action does not appear to be one of vasodilatation and no constant effect on coronary circulation in the experimental animal has been observed The drug has sedative and anesthetic properties and these probably account for its action It is inferior to the nitrites

***Alcohol*** *is an old remedy for angina still preferred by some who claim* that an ounce of whiskey or brandy has a speedier (and more satisfying) action The anginal attack vanishes as a sensation of warmth is produced by a dilation of the skin vessels The use of alcohol should be governed of course, by the frequency of the seizures experienced by the patient

**Routine Medication** The next question that arises after measures have been instituted to control the anginal attacks concerns routine medication Is there a drug that can be safely given over a long period of time that can be depended upon to decrease the frequency of the seizures? Again we meet the problem of the coronary dilator drugs and their efficiency Following appropriate changes in the patient s daily regime it has become the habit to prescribe one of the xanthine group of drugs I prefer theobromine sodium acetate in 0 3 Gm (5 grains) capsules after meals If after a trial of ten days the patient feels that the addition of this preparation has helped to decrease the number of attacks I continue it for a longer period If any symptoms arise from its use (nausea nervousness headache flatulence) it should be discontinued at once

While recently reviewing some clinic records of patients suffering from uncomplicated angina I found that the most popular prescriptions for these ambulatory cases are those containing one of the barbital preparations Elixir of phenobarbital with the occasional use of a purine derivative for a week or so and with nitroglycerine handy for attacks made up the regime of therapy in a large percentage of the cases A few physicians I discovered still cling to iodide medication supported by nitroglycerine when attacks occur Some patients in whom there is no reason to suspec syphilitic infection demand their drops at each clinic visit stating that the attacks are much worse when the iodide is discontinued The tremendous psychic factor present in many of the angina cases should not be overlooked when we attempt to evaluate this statement However there is no doubt about the fact that iodides in small doses over a long period *are less harmful than continued uncontrolled dosing with barbital preparations*

I have not used any of the various tissue extracts in the treatment of angina The nature of the substances my dislike of any form of injection treatment that brings the same group of patients back on the same days of the week and the lack of adequate controls evident in the literature have discouraged me at the start Other features of the treatment of angina that require fewer trips to the office are much more important Again a series of injections may act as a tremendous psychic stimulus to one group of patients making their evaluation difficult, if not impossible, while the

same treatment may cause another group to become weary of the physician and his regime The last group will end their visits and either continue without medical care or turn for treatment to one of the various cults The patients who continue treatment naturally make up in time a formidable group of cures but they are not representative of coronary disease and angina in any community and the physician by his over enthusiastic embrace of one method of therapy loses the opportunity of following many interesting cases The occasional patients who have unfortunately read about the value of the heart hormones in the daily papers are usually satisfied with the explanation of the status of this form of therapy

Digitalis is contraindicated in patients with uncomplicated angina (page 272) In some patients attacks may be increased in frequency following its use In the presence of congestive failure or auricular fibrillation that calls for digitalis anginal attacks, if formerly present commonly disappear

The physiotherapeutic measures of value in the treatment of angina will be found in Chapter 19

ABDOMINAL BELT
(See page 252)

ALCOHOL INJECTIONS
(See page 256)

EFFECTS OF AIR TRAVEL
(See page 250)

## ILLUSTRATIVE CASES

### CORONARY ARTERIOSCLEROSIS—MANAGEMENT OF ANGINA PECTORIS—THE QUESTION OF AIR TRAVEL

**Case 36** D C an advertising manager of 43 was first seen in June 1939 on which occasion the chief complaint was pain in the chest of a month s duration The pain was constricting in character always induced by exertion and relieved by rest nitroglycerine or whiskey No other symptoms referable to the cardiovascular system were present

THE PHYSICAL EXAMINATION showed nothing of significance B P 130/80 The heart was not enlarged The electrocardiogram however showed prolongation of the P R intervals When repeated after atropine the result was the same The Wassermann was negative and the blood count was normal

CLINICAL DIAGNOSIS A Etiologic Arteriosclerosis B Anatomic No cardiac enlargement *Coronary sclerosis* C *Physiologic Anginal syndrome First stage heart block* D Functional Classification Class 2 Therapeutic Classification Class C

**Discussion** In this case the diagnosis was made from the history alone The continued prolongation of the A V conduction time may be viewed in the light of a decreased blood supply to the bundle of His There may at one time have been an occlusion of one of the small branches of the right coronary artery with the production of a small infarct involving the septum The past history however failed to reveal any acute episodes of

chest pain that might have been caused by an occlusion However, the conduction defect produced no symptoms in this patient, and it was disregarded in planning the management

A review of this patient s daily regime showed habits of living that did not contribute to a successful control of the anginal seizures He was an executive in a large industrial concern and the nature of his position required him to spend most of his time traveling between branch offices in different parts of the country Since the distances were great air travel was almost always necessary This brings before us the question of permitting cardiac patients to be passengers on commercial air lines either for pleasure or when this method of transportation is essential for efficiency in business

Effects of Air Travel and Altitude on Patients with Heart Disease This problem has not been satisfactorily settled and will always have to be solved by a review of the circumstances present in each case A few general rules guiding this decision may, however, be stated The important factors that have a bearing on the decision we give are the cardiac status of the patient the distance of the flight contemplated the probable altitude and the previous experience of the patient with this form of travel During the past few years I have covered the entire route of several of the larger airlines in this country and have had first hand opportunity to observe passengers on large planes of the transcontinental type They are usually men in middle life or past middle life when the type of heart disease present is most apt to be coronary or hypertensive It is probably not a coincidence that air travel has a greater appeal to patients from these groups Speed and aggression become habitual and the air liner satisfies a compelling urge

Although close attention is paid to the physical condition of pilots and attendants on air lines there is little or no consideration given to the possible state of health of the passengers If this question arises at all it is referred to the family physician who renders the decision after an examination of the cardiovascular system However when we consider the number of passengers on air lines today and the few circulatory signs that are apparent on long distance flights the situation does not appear alarming Nevertheless there are exceptions and a few matters should be given careful consideration before granting the cardiac patient permission to travel by air

In the first place the anoxemia and the possible symptoms that may follow if the patient is kept for a long time at high altitudes in transcontinental flights are by no means negligible The possible effect of air sickness on the circulatory apparatus must also be weighed since the incidence of this condition is great particularly when flying at low altitudes It is also well to ascertain if possible the effect of previous flights on the emotions If there has been no previous experience on which to render a decision the possible emotional upsets that may occur particu

larly in bad weather should be given due consideration if the patient is known to have angina

Commercial air lines in the United States take passengers to altitudes varying between 4 000 to 10 000 feet During flight the passenger rests (weather permitting) in a very comfortable reclining chair The situation is totally different and consequently cannot be fairly compared to walking at a similar altitude With the patient resting comfortably altitudes reached by the plane have little effect on the normal circulatory apparatus Even at maximum elevations especially at night when the air is smooth and cool the effect may be most pleasant and even conducive to sleep Considering the number of older people who have all degrees of insufficiency of the coronary circulation if anoxemia had an extremely adverse effect we would hear about more fatalities during flight As the matter stands one air line reports but one fatality in flight due to cardiac failure in over 775 000 passengers Even this one accident was not believed to have been entirely caused by flying since the same incidence may occur in a similar number of persons selected at random on the ground

On the other hand in patients with slight or easily induced congestive failure the anoxemia of high altitudes is not recommended While it is entirely safe for the well-compensated cardiac patient to sit or recline at a maximum altitude of 10 000 feet for a period not exceeding three hours it is unwise for the patient who has the slightest sign of congestive failure to assume this risk The same applies to patients with advanced degrees of coronary insufficiency who have severe angina While anoxemia may not be the factor in inducing attacks excitement and fear likewise enter into the picture and must be considered These patients should not be permitted to travel by air

Only a few studies have been carried out on the effect of different altitudes The majority of these seem to show a slight elevation of the pulse rate a maintained systolic blood pressure a drop in the diastolic level and an increase in the pulse pressure at high altitudes Increase in the respiratory rate at high altitudes may deplete the blood of carbon dioxide and if continued could produce a transient alkalosis Stratosphere and sub stratosphere flying particularly in transcontinental trips will bring the problem of anoxemia and its effect on the cardiac patient very much into the limelight in the future No doubt engineers and physiologists working together will soon devise a method of maintaining a uniform oxygen content in the plane with automatic adjustment to altitude

To summarize I believe that all patients with any of the signs of congestive failure of recent origin or of long standing and patients who are subject to frequent attacks of angina should not be permitted to attempt flights of any distance This will include all class three and four patients (new classification) Patients in class two who suffer from advanced heart disease of any type and who experience dyspnea on slight effort should not be allowed to travel by air in rough weather or to take long transcontinental

flights They should also limit their flying to lines that do not go above 5,000 to 6 000 feet Class one patients after a short trial flight to allow the personal factors to be evaluated properly may be permitted to plan any trips by air provided that they restrict their flying to the large commercial lines

The patient under discussion was advised to accept another position with his firm at a slightly lower salary but one that did not entail long flights at frequent intervals His treatment was begun by a rest period of three weeks which was spent at the seashore During this time he was given triturates of nitroglycerine to take when needed for pain Routine medication consisted of a capsule containing phenobarbital 30 mg (½ grain), and theobromine sodium acetate 0 3 Gm (5 grains) after meals At the end of three weeks he was much improved and although the anginal pain was still present the attacks were not as frequent and were much less severe When re examined six months later the patient reported marked improvement in his condition Less tension was present in the new office, and there were greater intervals between attacks The only medication he was taking at this time was one nitroglycerine tablet as required

### Coronary Disease with Angina of Long Duration—Relieved by Abdominal Belt

**Case 37** Mrs R L a housewife of 65 was first seen in April 1935 complaining of pain in the left chest on exertion



Fig 103 Roentgen film showing cardiac enlargement of the hypertensive type Note enlargement of left ventricle Patient wearing belt

History Six years before the date of the first examination the patient began to have typical anginal seizures recurring frequently on exertion in cold weather and after heavy meals The attacks were relieved by rest or nitroglycerine

Physical Examination B P 110/80 Underweight Prominent abdomen The

rhythm was regular except for an occasional premature beat The heart was slightly enlarged (Fig 103) and a systolic murmur was heard over the apex and in the aortic area The aortic second sound was accentuated

LABORATORY DATA Wassermann reaction negative The electrocardiogram showed a left axis deviation The other features were normal Urine and blood count were normal

CLINICAL DIAGNOSIS A Etiologic Arteriosclerosis B Anatomic Slight cardiac enlargement Relative mitral insufficiency Coronary sclerosis C Physiologic Anginal syndrome Normal sinus rhythm D Functional Classification Class 3 Therapeutic Classification Class D

DISCUSSION This patient used more nitroglycerine tablets for the relief of her anginal attacks than any patient that I have ever observed Daily averages of 36 triturates (1/100 grain) were not unusual during winter months No untoward effects were noted

Attacks of anginal pain occurred after meals on slight exertion on going outdoors on cold days and invariably followed emotional disturbances It was evident that some other form of treatment that would give additional relief would have to be sought I had previously used an abdominal belt of the type recommended by Kerr[188] with indifferent results in a small series of anginal cases However since the patient while not overweight had a prominent abdomen I decided to try again the effect on the anginal pain The belt was first applied in June 1939 when the patient was leaving for the seashore Three weeks later she returned very enthusiastic and stated that she had been able to walk the entire distance from her bungalow to the beach without stopping a distance of one half a mile In addition to this she was able to take other long walks at the shore without pain or dyspnea for the first time in years The number of nitroglycerine tablets required showed a sharp decrease during this period She continued to take a tablet or two after meals but this she admitted was more because of habit than actual pain

Many of the patients successfully treated by Kerr were overweight and had dyspnea in the upright position that was relieved in recumbency Viewed under the fluoroscope in the upright position the diaphragm was found to be one or two interspaces below the normal position and its movements were restricted In the recumbent position however a much greater diaphragmatic excursion was noted Kerr concluded that the abdominal viscera together with the increased accumulation of fat serve as a counter weight suspended from the diaphragm The belt restores the function of the diaphragm and aids in the return of the blood to the heart This promotes more adequate filling of the coronary vessels and accounts for the relief of the anginal pain

Kerr supplements the use of the abdominal belt with dietary restriction He hopes that this method of promoting more adequate filling of the heart might in some measure prevent or postpone coronary thrombosis Abdominal belts are also advised for sedentary workers chiefly males who are overweight in the hope of avoiding symptoms of insufficiency of the coronary circulation

So far I have not had uniformly good results following the use of the abdominal belt of the Kerr type in patients suffering from angina In this patient the application of the belt brought about prompt and marked relief For this reason I believe that my series of cases so far has been too small to be of value in giving a fair opinion in the matter No doubt my selection of the type of patient most suited for the application of an abdominal belt has been poor Consequently the following case report and discussion will serve as a better illustration of this new method in the treatment of angina

ARTERIOSCLEROTIC HEART DISEASE COMPLICATED BY ANGINA PECTORIS SEVERE VERTIGO, AND POOR POSTURE—SUCCESSFULLY TREATED BY APPLICATION OF ABDOMINAL BELT (PRESENTED BY DR. WILLIAM J KERR*)

**Case 38** J D a 61 year old white male was seen in the Medical Clinic to which he had come because of progressively severe precordial pain of eight years' duration The pain radiated to the left shoulder and down the left arm was precipitated by exertion and occasionally by excitement He had been seen repeatedly and all the usual measures for the relief of angina pectoris including the nitrites xanthines and extreme regulation of his daily routine had been tried without avail He obtained relief from any single attack of pain by taking nitroglycerine He had been digitalized because of mild congestive failure three years previously For six months prior to the present admission to the clinic he noticed progressive exertional dyspnea and severe progressive vertigo which was generally precipitated by his rising suddenly and changing his position quickly This also came on after he had been on his feet for a short time

PHYSICAL EXAMINATION revealed a well developed slightly obese, adult white male with a drawn haggard facial expression appearing to be chronically ill Relevant physical findings were as follows

His posture was characterized by exaggeration of the cervical thoracic and lumbar spinal curves The ribs were fixed in a slightly inspiratory position The head was thrust forward and the abdomen was protuberant and somewhat pendulous The blood pressure in a reclining position was 130/86 mm of mercury When the patient stood the pressure changed to 1[illegible]8/[illegible] (it was impossible to determine satisfactorily the diastolic pressure) The heart was borderline in size on percussion with moderately good heart sounds The lung fields were clear Physical examination was otherwise essentially negative

The electrocardiogram revealed evidence of myocardial damage compatible with coronary occlusive disease The circulation time in the standing position was 32 seconds without abdominal support Following the application of an abdominal belt, the circulation time changed to 21[illegible] seconds The blood pressure stabilized at 13[illegible]/86 mm of mercury with the patient standing and wearing an abdominal belt Without support the patient was able to walk only ten yards at a military pace before the onset of anginal pain With a belt on he was able to travel 190 yards at the same pace without pain These determinations were made within five minutes of each other Studies in tidal air revealed an appreciable increase in the volume per respiration with abdominal support Fluoroscopy showed a maximum diaphragmatic excursion of 1 5 cm in quiet respiration With application of abdominal support the movement of the diaphragm increased to 3 cm The heart also seemed to decrease its amplitude of contraction with the patient's abdomen supported

This patient has been followed for six months during which time he has had only three or four attacks of angina These were all precipitated by excessive and ill advised exertion He is now able to carry on ordinary activity with perfect freedom from pain In addition he noticed instantaneous relief from his vertigo and this symptom has not returned His symptoms can be reprecipitated by removing his belt and subjecting him

* Professor of Medicine University of California Medical School

to exercise It has n t been neces ary for him to use nitroglycerin at all since his abdomen was properly supported

## Angina Pectoris in a Child Secondary to Aortic Regurgitation of Rheumatic Origin

**Case 39** S P a colored girl of 1 was admitted to the Ph ladelphia General Hospital in January 1930 complaining of shortness of breath and attacks of pain in the region of the h art that radiated to the left arm At the age of 10 the patient had t vo attacks of rheumatic fever with severe joint manifestations A year before admission there was palpitation and dyspn a on exertion Nose bleeds were fr quent A month b fore ad mis ion chest pain was complained of following moderate exertion or excite ment These attacks rapidly increased in severity and frequency

PHYSICAL EXAMINATION showed B P 140/40 Corrigan puls and marked pulsation of the vessels of the neck and arms The apex beat was in the anterior axillary line An ap cal diastolic thrill was palpable and presystolic and diastolic murmurs were heard over the mitral area The first heart sound was accentuated There was a loud diastolic murmur heard along the left sternal border

The electrocardiogram showed a left axis deviation and prominent P waves The T v aves were upright The Wassermann was negative Blood count showed Red blood cells 3 200 000 White blood cells 15 000 Hemoglobin 68 per cent (Sahli)

CLINICAL DIAGNOSIS A Etiologic Rheumati (inact ve) B Anatomic Cardiac enlargement Mitral stenos s mitral insufficiency Aortic insuffi iency C Physiologic Anginal syndrome D Functional Classification Class 3 Therapeutic Clas ificat on Class E

**Discussion** Angina pectoris in young people is rare When it occurs it is invariably associated with rheumatic heart disease and aortic regurgi tation White and Mudd[398] in 1927 collected 42 cases in patients under 30 years of age Stolkind in 1928 listed 29 cases Korns[194] and Levin [16] have reported single cases

Chest pain in young people associated with effort syndrome acute peri carditis or cardiac hypertrophy should not be confused with the type possessing the typical features of angina True anginal pain is sharper usually radiating to the left arm shoulder or left side of the face and is relieved by rest and nitroglycerine Exertion may show less direct rela tionship to pain production in young people than is usually the case in later years This child had attacks when at complete bed rest many came on during the night all were relieved by nitroglycerine

Many times in the presence of a free aortic leak angin i occurs without any evidence of obstruction to the coronary flow that can be grossly demonstrated The low diastolic pressure that accompanies aortic regurgi tation has been stated as a likely cause of anginal pain However Hochrein has shown *that the blood flow through the coronary arteries is modified* chiefly by the work of the heart rather than by variations in the aortic perfusion pressure Laplace has also demonstrated that the incidence of angina in aortic valvular disease is not definitely related to the height of the diastolic pressure [0]

The prognosis of angina pectoris in younger patients depends on the degree of cardiac involvement and the complications of the rheumatic state and differs in this respect from the angina encountered later in life The

type of therapy that offers the most relief of the angina in young people for the longest period is sympathetic nerve block

PARAVERTEBRAL ALCOHOL INJECTION OF THE SYMPATHETICS IN THE TREATMENT OF ANGINA PECTORIS (Discussion by Dr C A Steiner*)

In 1916 Jonnesco[175] first treated angina pectoris by sympathectomy and recommended removal of the middle and lower cervical and the upper dorsal sympathetic ganglia Later various modifications of this procedure were devised by Leriche and Fontaine,[14] Coffey and Brown[69] White[391] and others

Paravertebral alcohol injections to relieve the pain of angina were first used in 1926 by Swetlow[368] who injected the upper dorsal ganglia The greater safety of this method has caused it largely to replace sympathectomy in the treatment of angina pectoris

It is assumed that the pain of angina pectoris originates in the heart itself and is transmitted from there to the central nervous system The better understanding of this method of transmission is mainly responsible for the greater degree of success obtained by surgical procedures today than was formerly possible

White[391] states that

> afferent impulses giving rise to the sensation of cardiac pain leave the heart by cardiac sympathetic nerve fibers coursing to the cervical and upper dorsal sympathetic ganglia but they all pass to the spinal cord through the rami communications of the upper five thoracic nerve roots as through the neck of a bottle

(Fig 104) Since these impulses may best be interrupted at the upper dorsal communicant rami or their ganglia the surgical attack is directed toward these levels

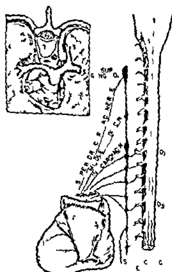
Obviously surgical measures must not be considered in the average patient suffering from angina pectoris whose symptoms are readily controlled by any of the forms of medical regime that have just been outlined They are however worthy of consideration in cases like this child where no response to adequate conservative treatment is evident, and where the frequent recurrence of severe attacks of pain in spite of medical treatment results in total disability Although dorsal sympathectomy is indicated where the risk is especially good alcohol injection is the procedure of choice in most instances No case need be denied the benefit of the latter procedure because of the severity of the cardiac lesion since the patients are not greatly disturbed by the injections

The purpose of paravertebral alcohol injections is to deposit a small quantity of 95 per cent alcohol in the immediate vicinity of each of the upper five thoracic sympathetic ganglia or their rami communicantes The destructive action of the alcohol produces a lasting interruption of the nerves carrying the painful stimuli from the heart

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Some idea of the nature of this procedure may be obtained from the following brief resume of the technic developed by James White[491] (Fig 105)

The patient lies on his side in his own bed with the head flexed and the knees drawn up The spine should be straight The bony landmarks are the spinous processes of the vertebrae the tip of each marking the level of the transverse process and posterior angle of the rib below After preparing the back a mark is made 3 to 4 cm directly lateral to each spine from the seventh cervical to the fourth dorsal vertebra Needles 8 to 10



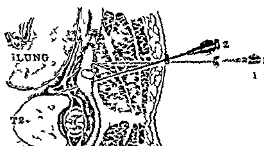




Fig 104 Diagram of the sympathetic nerves of the heart The direct thoracic cardiac nerves are shown joining the second third fourth and fifth thoracic ganglia with the posterior cardiac plexus (From The Autonomic Nervous System J C White New York Macmillan Company 1935)

Fig 105 Paravertebral injection of thoracic sympathetic ganglia Method of inserting needles (*Ibid*)

cm long are inserted perpendicular to the back to a depth of 2 to 5 cm at which point they should be in contact with the transverse processes or ribs The lower borders of the ribs are determined the needles inclined slightly in a caudal direction and further inserted at an angle of about 20 degree toward the midline until bone is again felt approximately 3 cm beneath the ribs The needles should then be in contact with the lateral aspect of the vertebrae or the heads of the corresponding ribs

> The sympathetic trunk lies at this depth running along the anterolateral aspects of the vertebrae and looping over the heads of the ribs Novocaine injected in this region will diffuse freely

through the retroperitoneal areolar tissue infiltrating the spinal nerves the communicant sympathetic rami and the gangliomated chain[391]

Making sure that the tip of the needle has not penetrated the pleural cavity a blood vessel or the subarachnoid space 2 cc of a 2 per cent novocaine adrenalin solution is injected through each needle Within 15 minutes characteristic signs of intercostal and sympathetic nerve paralysis (axillary anesthesia warmth of the arm etc ) should appear Then to insure complete anesthesia 2 or 3 cc of a 1 per cent novocaine solution is injected into each needle, followed by the slow instillation of 5 cc of 95 per cent alcohol

The patient should remain in the same position for at least an hour to allow the alcohol to become fixed in the tissues Usually the patient may get up on the following day and leave the hospital in three days

Sympathectomy is seldom used since alcohol injection can be performed with almost equal success and with much less risk However, when it is used the procedure of choice at present is the removal of the first second and third thoracic ganglia through a posterior approach, resecting the second rib

Obviously paravertebral alcohol injection can be safely and successfully carried out only by an operator who has had considerable previous training first in the anatomic laboratory and then as an assistant to a surgeon skilled in the method described above

The most troublesome complication following alcohol injection for the relief of angina is a painful intercostal neuritis which may persist for several months If the patient has obtained relief from the anginal seizures however this is seldom a matter of much consequence to him If the needle should accidentally pierce the pleura during its insertion pneumothorax may result but is rarely dangerous if promptly diagnosed and treated There is practically no mortality from the injection procedure itself, and even sympathectomy carries with it a very low risk if reserved for selected cases

Keeping in mind the fact that the cases of angina pectoris requiring surgery are usually the most serious ones the results are extremely good From the reports of James White[391] Paul White[393] Smithwick[illegible] Swetlow[368] Pletnev and Hesine[illegible] and others it appears that excellent results may be expected in about two thirds of the cases and marked improvement in about one half of the rest The relief obtained from either alcohol injection or sympathectomy is usually permanent

# 8

# CORONARY ARTERY DISEASE INCLUDING THROMBOSIS

This strange disease of modern life —MATHEW ARNOLD *The Scholar Gypsy* Stanza 21

Angina pectoris and coronary thrombosis are both associated with coronary artery disease The former is a functional condition in which the blood supply to a section of cardiac muscle is temporarily deficient while

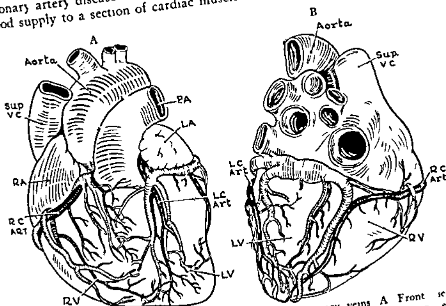



FIG 106 The great vessels coronary arteries and coronary veins A Front se B Base and diaphragmatic surface P A pulmonary artery Sup v c superior vena cava; R A right auricle L A left auricle; R C Art right coronary artery R V right ventricle L V left ventricle; L C Art left coronary artery

the latter is an organic lesion where the blood flow is permanently blocked by a clot and degenerative changes in the cardiac muscle ensue These dramatic episodes have focused a great deal of deserved attention in recent years on this typically American disease and a number of important advances have been made Electrocardiography has progressed to a point where not only is the diagnosis of occlusion of a coronary artery possible in 95 per cent of the instances but the site of the infarct can also

be accurately determined As we view our increasing knowledge with satisfaction many can still recall the day when thrombosis in the coronary tree was merely an interesting necropsy revelation Following Herrick s description in 1912 the importance frequency and clinical features of acute occlusion were established, and the modern physician has become skilled in its detection

In a series of 2877 consecutive autopsy reports studied by Levy [4] lesions of the coronary arteries were found in 25 9 per cent In half of these cases the involvement was slight or moderate causing no impairment of the coronary blood flow These autopsies covered a 22 year period and show a slight but steady increase in the incidence of the disease However it is most significant that diagnoses based on clinical observations alone have shown a greater increase in all clinics White [393] studying a group of 2 314 patients with heart disease in New England reports that 37 per cent were diagnosed coronary disease It is evident then that the diagnosis is much more often made during life than at autopsy in other words we are today acutely aware of the possibility of coronary disease, and its clinical signs and symptoms are seldom missed

## ETIOLOGY

Coronary artery disease causes cardiac damage by reducing the blood supply This may occur as a slow insidious process not reaching a degree sufficient to cause symptoms until late in life, or it may occur with dramatic suddenness at a much earlier age

**Arteriosclerosis** By far the most common etiologic factor in producing changes in the coronary tree is arteriosclerosis (Fig 107) It was present in over 95 per cent of Levy s series while syphilis was responsible in only a small percentage of the cases Much rarer causes of impairment of the blood supply to the myocardium are coronary embolism periarteritis nodosa and rheumatic coronary arteritis

The common arteriosclerotic lesion consists of abnormal thickening of the elements making up the intima of the vessel Areas of atheroma soon develop in many cases and these may soften rupture, and discharge their contents The rough areas that remain become suitable spots for the deposition of fibrin from the blood stream and a start is made for a thrombus which may rapidly develop where the vascular bed is narrowed and the circulation slowed

Inquiry into the cause of these fatal events in the coronary circulation would again lead us into a discussion of the theories that have been advanced to explain arteriosclerosis Suffice it to say that many times the stress and strain of modern life are reflected in the coronary tree Hypertension if long continued exerts a harmful effect on the coronary arteries and the spot that bears the brunt of this burden and consequently the one apt to show the most change is the anterior descending branch of the

left coronary artery Hypertension may be influenced by environmental factors when the individual possesses the proper constitutional background In this event the tempo of modern life may after all have a decided influence on the speed with which coronary sclerosis develops Coronary accidents have replaced coma as the main causes of death among diabetics Here the endocrine imbalance resulting from the disturbance of the metabolism of fat and cholesterol plays a major role Finally the fact that we may inherit a tendency to early change in the structure of our arteries cannot be overlooked

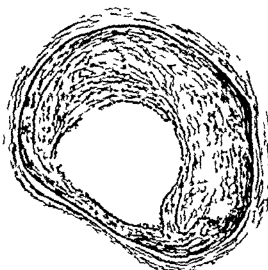


Fig 10 Atherosclerosis of a coronary artery

Syphilis has been shown to be no more frequent among patients with coronary disease than among those without it Syphilitic aortitis may involve the mouths of the coronaries and cause complete obstruction but damage of the coronary arteries themselves by syphilis is rare Likewise the part played by rheumatic infection in the production of coronary disease is obscure although we do know that involvement of the intima and media occasionally has been known to follow rheumatic infection with the subsequent formation of mycotic aneurysmal dilatations

The role of focal infections in the production of coronary disease is a much disputed and still unsettled point Likewise the relationship of coffee tea tobacco and alcohol to speeding the development of the sclerotic process cannot be said to be definitely established Emboli (air fat tumor cells fragments of vegetations) may occasionally invade the coronary arteries and produce infarction in patients of any age but this accident is rare

## INCIDENCE

**Age** Arteriosclerosis of the coronary tree sufficient to produce clinical symptoms is uncommon under 40 After this age the incidence increases with advancing years reaching its peak between the ages of 60 and 70 Bland and White[396] have shown that coronary thrombosis with infarction occurs at a younger age than coronary disease as a whole The highest incidence of acute coronary thrombosis in their series occurred between 50 and 60 years of age

**Sex** Coronary disease is much more frequent among men than among women All statistical studies confirm this statement some reporting the ratio to be as high as seven to one Occupation may have a direct relationship to this predominance of the male since the overweight over worked, overactive business or professional man of today is often the victim of coronary disease Levy s studies have revealed that the largest percentage of cases of coronary sclerosis occur among foremen and skilled workers with the professional and executive group taking second place

## ANATOMY AND PATHOLOGY

An understanding of certain fundamental anatomic and pathologic facts concerning the coronary tree is essential to our discussion In the first place the coronary lesion may be microscopic capable of producing no great reduction in the blood flow to the heart muscle consequently no structural damage is evident and no clinical symptoms appear However if more marked narrowing occurs and thrombosis follows the outcome will depend on the size of the artery occluded and on the extent and number of anastomotic branches that are present These branches vary in different patients and at different periods of life in the same patient As we grow older the number of anastomoses in the coronary circulation increases preparing us to withstand the accidents that may attend advancing years If the process of narrowing is a more gradual one these collateral pathways may develop to a remarkable degree thus increasing the chances of survival when the occlusion finally becomes complete

Sudden occlusions are not infrequent occurrences in which crises all the cardiac reserve must be summoned for survival In these emergencies in addition to the collateral channels we have referred to some circulation may be maintained through the thebesian vessels which are small channels communicating directly with the interior of the heart Blood vessels present in normal attachments of the pericardium likewise may be called upon to act as collateral channels In emergencies even the small blood vessels in adhesions which may be present and bridge the pericardial sac often dilate and come to the aid of a failing coronary circulation The Beck operation is based on the assumption that a transplant or

a bridge of pectoral muscle may be effective by augmenting the coronary supply in this manner

## DIAGNOSIS

It now becomes evident that the symptoms present in each patient will depend largely upon a number of variable factors and cannot be expected to be the same in every instance Occlusion of small arteries may take place with slight subjective manifestations It is quite likely that many times the incident is entirely overlooked since the cardiac balance is either little disturbed or quickly restored If a large artery is involved however, the clinical picture will be typical and the diagnosis may be made on inspection Between these two extremes the latent case on the one hand and the patient with a large occlusion on the other all gradations may be encountered Irrespective of the size of the infarct a great deal depends on the previous condition of the heart and whether or not a serious cardiac arrhythmia is precipitated by the accident

It is by no means an easy task to diagnose the latent case Subjective and objective evidence of the progress of the sclerosis may be entirely absent until thrombosis suddenly takes place It is likewise not uncommon to discover a markedly advanced coronary sclerosis at autopsy in a patient who died of some other disease If the coronary sclerosis advances slowly narrowing and finally obliterating many small end arteries the resulting infarcts heal and become invaded by fibrous tissue The myocardium then becomes progressively weaker and congestive failure may supervene and at autopsy the heart will show dilatation and much myocardial fibrosis Is this the heart lesion that we have been in the habit of referring to as chronic myocarditis If so it should be viewed in a different light since the fibrous tissue does not have its origin in an inflammatory process but is the end result of arteriosclerosis of the coronary arteries The cardiac dilatation that occurs usually causes stretching of the mitral ring before death and the systolic apical murmur of relative mitral regurgitation is frequently heard More advanced cases may show sclerosis of the valvular structures and the regurgitation may then be a combination of these two factors

**Cardiac failure** may be gradual in its onset As the blood supply to the heart muscle is slowly curtailed dyspnea appears on less exertion Edema of the feet may be present at night and finally attacks of cardiac asthma or cardiac dyspnea give evidence of a diminished myocardial reserve Pulmonary edema congestion of the liver and other signs may be added to the picture at this stage

**Cardiac Irregularities** The small infarcts in the myocardium that undergo healing may act as irritable foci and their presence may then be made known by the occurrence of the frequent premature beats that interrupt the cardiac rhythm Paroxysms of tachycardia from a succession of these stimuli may be precipitated by a sudden occlusion The onset of

chronic auricular fibrillation is not uncommon as the involvement of the auricular muscle advances Occlusion, gradual or sudden of the artery that supplies the bundle of His may cause delay in the passage of the impulse for cardiac contraction from auricle to ventricle (Case 36) Sudden occlusion of this artery may cause complete heart block I have seen Adams Stokes seizures occur following an attack of chest pain and a drop in the ventricular rate to 40 Pulsus alternans may be evident in patients with a sudden thrombosis or in cases where chronic failure has progressed to its terminal stages and the ventricular muscle is making its final struggle to survive by partitioning its remaining reserve strength

**Gastro intestinal Symptoms** Sometimes as Riesman has shown[314] the symptoms produced by advancing sclerosis of the coronary arteries may be entirely gastro-intestinal This is not surprising when we consider the fact that the vagus nerve supplies both the heart and the alimentary tract Consequently abnormal states of the heart may be reflected in the group of symptoms often referred to by the patient as indigestion' A similar reflex from the stomach to the coronary tree may be possible this pathway may account for attacks of pain or even occlusions that occur too frequently after eating to be merely co incidental Many patients who reflect the signs of advancing heart disease in chronic gastro intestinal complaints are often treated for gallbladder disease (page 479) Even the pain of an acute occlusion may have an abdominal reference and if attended by nausea and vomiting the differential diagnosis may be difficult (Case 101) It is small wonder that many attacks of coronary occlusion masquerade under the term acute indigestion

**Chest Pain** As the coronary lesion progresses a prominent symptom is chest pain it occurs usually in paroxysms and is the direct result of myocardial ischemia This cardiac pain may come on after emotion or excitement but is nearly always induced by exertion particularly when the patient hurries up a slight grade It is also accentuated by cold weather and by overeating The pain may be of the constricting type and remain confined to the front of the chest or it may radiate to the left arm both arms the neck or the jaw bone When it travels down the arm it is usually on the ulnar side and is felt in the ring and little fingers A reverse direction of the pain may be met in some patients in which event it starts in the arm or elbow and radiates toward the shoulder The intensity of the pain of angina varies according to the individual It may be quite typical in onset and distribution in some while in others it may be expressed as a slight ache in a tooth a finger or an elbow Nevertheless the pain carries the same significance in all Some patients will describe the sensation that accompanies narrowing of the coronary arteries as a constriction or tightening and will object to the use of the term 'pain' However this in no way detracts from the seriousness of the symptom Cardiac pain nearly always plays a prominent role in the symptomatology of those whose daily activities are greatest particularly if these activities are attended by emotional strain An attack may last a few seconds to a few minutes and is usually

relieved or at least greatly improved on rest (For a more complete discussion of this symptom, see Chapter 7)

Thrombosis As sclerosis of the coronary vessels progresses thrombosis may occur (Fig 108) The signs and symptoms attending the occlusion of a large vessel by a thrombus are well known Pain usually appears suddenly and increases until it becomes agonizing in its intensity It is usually substernal but may radiate to the epigastrium and be followed by nausea and vomiting When the infarct is large the patient is extremely pale and has the appearance of one acutely if not mortally ill A cold sweat appears the blood pressure drops and the pulse may for a time be weak and almost imperceptible Often in patients where the lesion is one of long duration and anginal pain a daily occurrence the attack may not present the textbook picture of pain and collapse but may be ushered in by a paroxysm of dyspnea pulmonary edema or congestive failure symptoms that characterize advanced coronary sclerosis In other patients subject to angina, a thrombotic occlusion may occur following an increase in the number and severity of the anginal attacks The pain of the occlusion is more severe and longer in duration than the pain of angina and does not yield to nitroglycerin

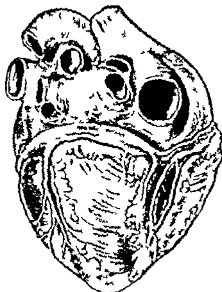


Fig 108 Posterior descending branch of the right coronary artery incised showing presence of an obstructive coagulum The diagnosis of thrombus was made by section

If the obstruction occurs in a vessel on the anterior surface of the heart, a friction rub may be heard over the localized area of pericarditis while if the infarct faces posteriorly this valuable sign is not elicited The absence of a precordial friction rub following acute chest pain however does not rule out an anterior occlusion

Since the infarct is in reality an area of necrotic material in the heart wall an elevation of temperature commonly follows its formation Leukocytosis likewise appears and the red blood cells show an increase in their rate of sedimentation

If the infarcted area extends to the inside of the ventricle the necrotic surface encourage the deposition of fibrin and a mural thrombus may appear parts of which may subsequently become dislodged and act as emboli Those originating in the right ventricle lodge in the lungs while

those dislodged from the left side of the heart (Fig 109) come to rest at points in the systemic circulation the brain, the spleen the kidney the



Fig 109 Mural thrombus at site of cardiac infarct (marked by arrow) (Autopsy No 3 390 Philadelphia General Hospital)

mesenteric vessels or the extremities Embolism is a frequent cause of sudden death following a coronary artery occlusion that is often overlooked It is more common than cardiac rupture When coronary occlusion

is suspected or when it occurs in the absence of pain and the possibility of a mural thrombus is not considered a primary brain or pulmonary lesion may be diagnosed Consequently embolic manifestations that suddenly appear in other organs of the body should always prompt us to investigate the integrity of the coronary tree

*Following acute occlusion* a number of cardiac irregularities may appear The most serious among these are paroxysmal ventricular tachycardia and ventricular fibrillation In many cases where large occlusions occur the immediate onset of ventricular fibrillation usually ends the picture

**Aneurysm** If the patient survives the initial shock of the coronary accident and the blood pressure again mounts all danger is not over Unless care is used in management a cardiac aneurysm which is merely a localized bulging of the heart wall may form at the site of the infarction (Fig 110) If the patient s activities are resumed too soon after a large occlusion rupture of the heart wall through the infarcted area may occur Any sudden exertion or strain during the period of bed rest may also produce the same result

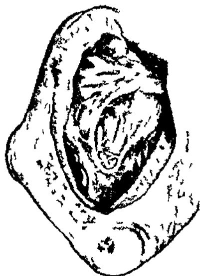


Fig 110 Cardiac aneurysms

This is the classical picture of coronary disease ending in thrombotic occlusion of branches of the coronary artery The symptoms point to the progress of the underlying lesion and the therapeutic implica tions are clear However not every case is so easily recognized Where little interference with normal coronary flow is produced symptoms may be entirely absent until the first occlusion suddenly appears The presence of coronary disease should be suspected in any patient over 40 who complains of dyspnea or chest pain particularly if this patient is an obese male A positive family history or the presence of hypertension should strengthen the suspicion Many times the electrocardiogram proves a valuable help if suggestive changes are seen On the other hand a negative electrocardiogram does not eliminate the possibility since minor alterations in the blood flow in the absence of acute myocardial infarction produce no marked abnormality If the patient is seen soon after the onset of a small occlusion a normal tracing may be obtained Consequently a repetition of the test in a few hours is advisable since this may show significant alterations (page 643) Usually any marked changes that occur in the features of the electrocardiogram from day to day in a person who gives enough of a history to arouse suspicion of the presence of a small occlusion may be attributed to

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this cause and are valuable in reaching a final diagnosis It is important to recognize the mild cases and establish proper treatment early Few of the *characteristic clinical signs such as low blood pressure, fever leukocytosis* and shock may be evident in which event the electrocardiogram assumes a place of great importance in diagnosis

Severe grades of anemia may produce chest pain as a result of myocardial anoxemia In most of these cases it is quite likely that some degree of coronary sclerosis already exists and the addition of the anemia is enough to precipitate symptoms of insufficiency In the same manner hyperthyroidism by increasing the metabolism may place just enough added strain on already diseased coronaries to produce attacks of anginal pain A similar strain on the coronary circulation may occur in older patients with the onset of paroxysms of tachycardia flutter or fibrillation

Levy has called attention to the production of chest pain simulating coronary occlusion that may occur in persons consuming large quantities of tea or coffee Tobacco may likewise aggravate the symptoms of coronary disease [49 399] Chest pain often accompanies effort syndrome but here the other features of the examination are usually sufficient to make the distinction

The chest pain and precordial friction that accompany acute fibrinous pericarditis are rarely confused with coronary thrombosis The age of the patient and the signs of infectious carditis may serve to make the distinction although the features of the electrocardiogram in the two instances may be quite similar (see Fig 252)

## PROGNOSIS

The prognosis of coronary disease is variable Many times the condition is discovered *in old people* at postmortem In direct contrast to the long symptom free existence possible in some of these cases there are instances in younger individuals where a thrombus forming on a single plaque in a large artery may spell quick disaster In between these two extremes there are numerous cases where the diagnosis may be made and successful treatment carried out over a long period Much of course depends on the patient s intelligence nervous temperament and willingness to co-operate Some apparently hopeless cases (page 278) may be carried successfully over a period of years with no increase in symptoms and in some instances even *an improvement in the exercise tolerance may be observed* Patients with syphilitic aortitis who have involvement of the coronary arteries at their site of origin do poorly and generally succumb at an early age Hyperthyroidism accentuates the symptoms of coronary disease but if successfully managed the outlook is good for after thyroidectomy the symptoms of coronary sclerosis may again retreat below the clinical horizon and remain out of the picture for years As a rule progress of the coronary lesion may be roughly gauged by the ease of production of anginal pain when this symptom is present Increasing narrowing of the coronaries is reflected in

the patient's complaint that the pain appears on less exertion Finally pain appearing on the slightest exertion or while at rest suggests an advanced lesion and a poor prognosis ı Some patients following a typical history of angina may have an occlusion and subsequently when ordinary activity is again attempted have no reappearance of anginal pain (see Case 40) Here we can infer that the artery occluded and the area of muscle it supplied were the only sources of painful afferent stimuli arising from the heart Prognosis in these cases should be excellent

Occasionally autopsy reveals only a very small coronary occlusion that appears insufficient to account for the fatal outcome In these cases death probably results from the reflex spasm produced in neighboring coronary vessels by the occlusion This increases the area of ischemia and precipitates fatal ventricular fibrillation Reflexes from the gastrointestinal tract following a heavy meal may also produce similar fatal spasms of coronary branches Consequently we cannot say that all small occlusions in their early stages are invariably associated with good prognoses



Fig 111 Atheroma of the abdominal aorta

While the occurrence of coronary thrombosis is a serious complication in coronary disease it does not necessarily lead to a poor prognosis White has reported several instances of long survival following a proved occlusion One patient[394] survived 24 years and another 17½ years

## TREATMENT

### General Principles

The objects of therapy in coronary disease are to relieve the pain of angina or occlusion and to establish a satisfactory daily regime in order to postpone for as long as possible heart failure either of the congestive or

coronary type As we shall see in the discussion of the treatment of the cases that follow each patient presents problems demanding individual attention Coronary disease is chronic and slowly progressive There is no specific regime of therapy unless the involvement is secondary to syphilis The physician therefore meets all the handicaps and situations that occur in the management of any patient of this age group who has a chronic disease

At the start the patient must be willing to accept certain restrictions in his activity in return for a less demonstrative coronary circulation No difficulty may be encountered if the patient s confidence is gained in the beginning by frankly stating the situation in simple terms and explaining the action of the few drugs prescribed This may be a time-consuming procedure but it pays dividends in the end for a skillful physician may keep his patient s outlook on life unchanged in the face of advancing lesions Every detail of the day s program should be reviewed from the time that the patient gets up in the morning until he goes to bed at night This should include what he eats where he eats and the time allowed in the process No happenings in the course of the day should be considered too trivial to be included in this review and weighed in the light of the possible effect they may have on the coronary circulation

The questions that the patient asks the physician will depend a great deal on the patient s station in life these questions should always be answered with this circumstance in mind Usually enough has been learned by the time the interview ends to guide the physician in his answers The patient s nearest relative should always be given full information in regard to the exact state of affairs and the fact should be emphasized that a patient with coronary disease and angina may live for years but sudden death at any time always remains a possibility

If the patient is free of symptoms but shows evidence on clinical or laboratory examination of the presence of coronary disease this rearrangement of the regime proper dietary management (page 541) and removal of obvious foci of infection constitute all the necessary measures The administration of iodide of potassium in doses of 5 to 10 grains (0 3 to 0 65 Gm ) three times a day appears to be a time honored procedure Some claim that this drug inhibits vascular spasm others that it inhibits the advance of the arteriosclerotic process that is favored by high cholesterol diets others that its action comes about through a change in the viscosity of the blood but most physicians when questioned are usually at a loss to explain its rationale If the Wassermann reaction is negative and hyperthyroidism is absent I fear that the iodides have gained most of their reputation by coming to the rescue when no other remedy appears definitely indicated

## DRUGS

**Xanthines** The use of the xanthines as dilators in disease of the coronary arteries is a valuable addition to our therapy Three alkaloids are

included in the caffeine group caffeine (trimethyl xanthine) theobromine and theophylline (dimethyl xanthine) all are purines and therefore closely related to uric acid These substances are insoluble in water but when combined with salts like sodium salicylate sodium acetate or ethylene diamine they become soluble and in this form acquire a wider range of clinical usefulness The most popular member of the group is theophylline ethylene diamine (aminophylline euphyllin or metaphyllin) In experimental work on laboratory animals most observers agree that the coronary arteries are dilated by the drugs of this group [104, 113, 120] Even when administered in great dilution to isolated hearts the coronary output increases 50 per cent Cushny suggests that the effect may follow a direct stimulation of the heart muscle However uniformity of opinion as to the effect of the xanthines in man is still lacking Some clinicians find them extremely useful in increasing the blood supply to the myocardium Experimental proof of this stand may be found in the work of Fowler and his associates[104] who administered aminophylline to normal dogs after ligation of a coronary branch and found that the resulting infarct was smaller than in control dogs where the drug was not used However it is only fair to state that similar results were not obtained by Gold and his workers Wiggers and Green likewise found the xanthine group ineffective in increasing the coronary flow in dogs after experimental coronary occlusion [119, 120]

Taking into consideration the variable factors that exist in every case of coronary disease in man a definite decision concerning the value of the xanthines is difficult to obtain While I do not doubt the good results reported in the literature by many observers I have not learned to depend on these drugs so much to relieve or prevent pain in coronary disease as I have for their diuretic action in cases of congestive failure and in the treatment of certain types of dyspnea where morphine is poorly tolerated

Another point against the wholesale use of the xanthines is the expense to the patient The best rule in practice is to administer one of the group for example theobromine sodium acetate in 0 3 Gm (5 grain) doses after meals for a period of a week If there is no definite improvement in the symptoms it may be discontinued Considering the rigid calcified coronaries that we see so often at autopsy I doubt the efficacy of these drugs as dilators in every case However they may be efficient in the treatment of coronary disease in younger patients where reflex spasm of the vessels still plays a major role

Barbiturates At the present time sedatives of the barbituric acid series are very popular in the treatment of coronary disease particularly when associated with hypertension While there is no doubt that these drugs serve to allay fear relieve insomnia and nervousness and by blocking reflex pathways reduce the number and severity of anginal attacks they should be used with care and their administration should be continuous only when the patient is under the constant supervision of the physician Untoward symptoms are not uncommon while in excessive doses these drugs can do harm It is wise not to exceed 15 to 30 mg (¼ to ½ grain)

of phenobarbital three times daily A mild sedative effect is usually obtained by this dose, which is valuable in quieting the heart and decreasing the incidence of ectopic rhythms

**Digitalis** Recent studie all seem to confirm the opinion that digitalis is of questionable benefit in cases of coronary disease in the absence of signs of congestive failure Ginsberg and his associates[116] studied the effect of various digitalis preparations on the coronary sinus outflow of heart lung preparations and of intact dogs They observed an initial decrease in the coronary flow lasting about ten minutes which was generally followed by an increase for the remainder of the experiment Variable results were obtained in intact animals many showing no change at all in the coronary flow following digitalis administration These carefully performed experiments indicate that the constrictor effect of digitalis upon coronary circulation is not sufficient to contraindicate its use unless there is present an extreme deficiency in the coronary flow Travell et al [375] studied the effect of digitalis on cats three weeks after experimental ligation of a coronary vessel and found all the animals were more susceptible to digitalis than normal controls About three fourths as much digitalis was needed after ligation of a coronary vessel to cause a ventricular ectopic rhythm and death As a rule they found that the larger the infarct, the more susceptible the animal appeared to be to the drug but many exceptions were noted These investigators concluded that digitalis administered after coronary occlusion at least in the experimental animal favors the production of abnormal impulses in the infarcted area that may lead to attacks of ventricular tachycardia and fibrillation In view of these results the best rule to follow in practice is to withhold digitalis in coronary di ease in the absence of signs of congestive failure With the appearance of cardiac decompensation however digitalis should always be given in sufficient amounts to achieve a therapeutic result (Chapter 2)

**Nitrites** should be used as required for the anginal pain that may complicate coronary disease Fresh nitroglycerine triturates (1/100 grain) dissolved under the tongue as occasion demands still constitute the best form of nitrite therapy The tablets should never be prescribed to be taken at stated intervals during the day Occasionally good results may be obtained by using a tablet just before an activity that previously provoked an anginal attack In this way it may be possible to open up some collateral channels in the coronary circulation by combining the dilating effect of the drug with the increased blood flow that follows the exertion A further discussion of nitrite therapy will be found in Chapter 7

## Management of Acute Occlusion

**Control of Pain** The first thought is to control the pain which is usually of a severe agonizing variety Consequently a hypodermic injection of morphine sulfate ¼ grain (15 mg) should be given at once Continuation of the pain calls for a similar dose in a half hour and in some cases several hypodermic injections may be required If possible the patient

should be made comfortable where he is found by the physician on his arrival for it is distinctly harmful to move a patient in this condition unless it is an absolute necessity The patient should not be undressed until some degree of recovery is evident in the appearance pulse and blood pressure The application of local heat is advisable in the form of a hot water bottle or electric pad At this stage it is unwise to use a multiplicity of restorative measures The acute insult to the myocardium contra indicates the use of large infusions that may add to the circulatory load It is also better policy to withhold adrenalin and ephedrine unless the emergency is extreme and the patient is unconscious and pulseless

**Absolute physical and mental rest** are essential for the patient and are usually obtainable only when tasks are found in some other location for the excess number of anxious relatives that usually crowd the scene It is far better to engage a capable attendant at the start The type of bed and other matters relating to the general care of the patient with failure of the coronary circulation do not differ from those recommended for failure of the congestive type (page 74)

Once the diagnosis is established too frequent examinations disturb the patient A trip to the hospital to secure an electrocardiogram or roentgenogram to prove what is obvious on clinical examination is likewise no great advantage at this stage of the treatment

When the pain has been completely controlled the hypodermics of morphine should be stopped Enough sedation should be given however to insure a good rest during the first few nights of the illness It is quite possible that the morphine in some patients may induce vomiting so it is wise to discontinue it as soon as the pain disappears and use as a substitute for allaying restlessness one of the barbituric acid derivatives or sodium bromide Sedatives are necessary in securing the relaxation that is so essential during the first few days following an acute attack of coronary thrombosis Since nausea and vomiting may accompany the state of shock that attends the accident or may follow the large doses of morphine the diet for the first day or two will not be a matter of great concern (Chapter 21)

**Constipation** usually follows the morphine but may be disregarded during the first two days unless a great deal of distention develops in which event an enema is indicated It is wise whenever possible to postpone the use of enemas until complete circulatory balance has been restored and the patient is definitely on the road to recovery Later in the course of treatment a routine laxative sufficient to secure one bowel movement daily should be given and the patient instructed to avoid straining at all times

**Oxygen** In many cases following occlusion of a large coronary branch cardiac action is much embarrassed and life hangs in the balance Survival in these instances will depend largely on the aid rendered by the physician and the speed with which he acts Often the administration of oxygen may prove the deciding factor in swinging the balance in favor of recovery

In patients who show marked dyspnea and cyanosis oxygen is best given in a concentration of 45 to 50 per cent using a tent but other methods if intelligently used should prove equally efficient (page 99) Oxygen brings prompt relief to the respiratory embarrassment and restlessness, diminishes or abolishes the cyanosis, and slows the cardiac and respiratory rates [14 17] Levy and Barach[1 3] recommend the use of an oxygen tent over long periods for patients suffering from acute attacks of coronary occlusion If the patient is removed from the tent too soon, the symptoms of circulatory distress may recur

**Glucose** If we continue to plan our treatment physiologically we should next attempt to make more glucose immediately available to the overtaxed heart muscle All laboratory workers have witnessed the prompt revival of the isolated heart of the experimental animal that follows the addition of glucose to the infusion entering the coronary vessels To produce some measure of the same effect is our hope when we inject small amounts of 50 per cent glucose solution As previously stated large intravenous injections are contraindicated since they increase the blood volume at a time when the heart is already overloaded consequently only small amounts of glucose should be used and all injections should be given slowly I prefer 50 cc of a 50 per cent glucose solution After warming the ampule to body temperature the technic may be simplified by drawing the entire contents into a large syringe and injecting it into an arm vein Five minutes should be allowed to inject 50 cc so that the danger of adding too quickly to the blood volume as well as that of venous thrombosis may be avoided Unfortunately the same effect is not obtained by giving a similar amount of glucose by mouth The excessively sweet solution may cause nausea and slow absorption from the gastro intestinal tract may be expected in the presence of so marked an impairment of the circulation The introduction of a hypertonic glucose solution into the circulation has a vasodilator effect on blood vessels, including the coronaries and this may account for some of the benefit derived from these injections

If respiratory difficulty is present in the form of Cheyne Stokes breathing or paroxysmal dyspnea (cardiac asthma) some improvement may be noted following injections of glucose Caffeine sodium benzoate may be useful in cases of acute occlusion if given intravenously in doses of 7½ grains (0 5 Gm ) every four hours

I have seen two patients (see Figs 243 and 244) develop sudden complete heart block shortly after the onset of an acute coronary occlusion Typical Stokes-Adams seizures occurred in one followed by death six hours later In the other patient these attacks did not occur normal rhythm was re established in two days and the remainder of the convalescence was smooth The management of Stokes Adams seizures is described on page 405

**Ventricular tachycardia** (page 390) may occur following an occlusion and precipitate cardiac failure The attack may end spontaneously but as soon as the diagnosis is established, quinidine sulfate should be given The usual initial dose should be 0 1 Gm (1½ to 2 grains) followed in

two hours by a slightly larger dose 0 2 Gm (3 grains) and later by 0 3 Gm (5 grains) every three hours Quinidine is usually well tolerated by patients suffering from acute occlusion When the attack is terminated it is well to continue 0 3 Gm (5 grains) of quinidine three times daily as a maintenance dose for a period of ten days to two weeks Levine [18, 19] recommends quinidine in 0 2 Gm (3 grains) doses three times a day for two weeks as a prophylactic measure in every case as soon as the diagnosis of coronary occlusion is made hoping in this way that such complications as paroxysmal ventricular tachycardia paroxysmal auricular and ventricular fibrillation may be prevented

Patients with cardiovascular syphilis may occasionally develop acute coronary thrombosis in which event the treatment for the acute episode is the same as outlined above When recovery is complete bismuth and the iodides may be started (page 214)

Complete rest in bed for six weeks is the prescription in all cases of acute occlusion While the practitioners of today are expert in the diagnosis of occlusion many allow their patients to be out of bed before the infarct has had time to heal firmly Willius has demonstrated that in 5 to 22 days after the formation of a coronary infarct connective tissue formation may be recognized and in four to six months the healing is complete The value of careful increase in exercise allowance after the initial six weeks of bed rest is therefore evident if ventricular aneurysms and other complications are to be avoided Useful guides to healing of the infarct are the behavior of the sedimentation rate (page 57) and the subsequent changes in the appearance of the electrocardiogram (page 636) As a rule return to full activity should not be allowed for three months

If at all possible much can be gained by a period of spa treatment at the time when the patient is first permitted to be out of bed (page 513) The education secured here in the matters of diet and activity often proves invaluable The patient learns to be satisfied with life on a much reduced plane which may be an important step toward prevention of recurrence of the episode

A great deal has been written concerning the use of tobacco by patients who have coronary disease Without reviewing the different sides of the question I believe that it is safe to state that enough is definitely known about the effect of tobacco in cases of arterial disease to prohibit its use by patients who are striving to live along with coronary artery disease and avoid its complications

SURGICAL TREATMENT

Newer treatments have a decided surgical trend Total thyroidectomy has been recommended for coronary disease as well for the complication of congestive failure This procedure has been discussed elsewhere (page 102) More recently Beck[96] has grafted vascularized tissues on the heart in the hope of providing the myocardium with an additional blood supply and relieving the anoxemia Skeletal muscle from the chest wall and fat from

the mediastinal and subcutaneous deposits are the tissues used in this operation. A graft from the left pectoralis major is applied to the surface of the heart which is approached from the left side of the sternum. At the same time the pericardium is roughened to encourage adhesions between the heart muscle and the pericardial fat that receives some of its blood supply from extracoronary sources. Powdered beef bone is used on the surface of the heart in the hope that the inflammatory reaction it produces may increase the number of vascularized adhesions between the graft and the coronary bed. Quinidine should be given routinely before and after this operation and the surgeon should be prepared to defibrillate the ventricles if ventricular fibrillation occurs at any step of the procedure (page 475). Patients are placed in oxygen tents for variable periods following operation. While Beck's mortality for the first 12 patients was 50 per cent, in the next nine cases the mortality was zero. So far the clinical results of this procedure are encouraging. Improvement in the patient's symptoms is brought about either by the increased blood supply or its more efficient distribution or by interruption at the time of operation of nerve pathways from the heart. Final opinion in the matter of the Beck operation will depend upon the clinical course of the first group of patients subjected to the operation when compared to a control series and the size of the collateral channels that will be revealed in the autopsy specimens.

At the present writing the physician should continue to treat his patients with coronary disease by the usual medical measures. I referred some patients for total thyroid ablation when this procedure reached the peak of its popularity but have so far referred none for the Beck operation. In the exceptional case where pain is extreme and cannot be controlled the paravertebral injection of alcohol (page 256) is the procedure of choice and is attended by less risk. However, like the nitrite the medical man administers, it is only directed toward relief of a symptom and does not strike at the defect responsible for the disease. The Beck operation, on the other hand, is planned to give the heart muscle more blood. If this is accomplished and the mortality remains low, our future choice in the matter is obvious.

### Management of Cardiac Complications in Diabetes Mellitus

*(See page 287)*

## ILLUSTRATIVE CASES

### Coronary Disease Complicated by Angina—Marked Improvement Following Initial Attack of Coronary Thrombosis

**Case 40** G. H., a city fireman of 46, when first seen on 10/3/33 complained of a crushing chest pain radiating to both arms, sudden in onset and increasing in severity. Milder attacks of pain similar in type had been present for one month, usually appearing when the patient walked up a long hill near his home and disappearing promptly after a short rest.

The past medical history was negative. *The family history showed that his father* and one brother died suddenly of heart disease.

PHYSICAL EXAMINATION BP 90/60 T 97 ? P 100 Ashen pallor dyspnea and sweating were noted No cardiac enlargement There was a systolic murmur over the aortic area transmitted to the vessels of the neck The lungs were clear Abdomen negative No edema

DIAGNOSIS A Etiologic Arteriosclerosis B Anatomic No cardiac enlargement Coronary occlusion Coronary infarction C Physiologic Anginal syndrome D Functional Classification Class 4 Therapeutic Classification Class E

**Discussion** An injection of morphine sulfate 15 mg (¼ grain) was sufficient to give complete relief of pain In two hours the circulatory equilibrium was restored the blood pressure rose to 110/80 the color improved and the dyspnea was no longer in evidence

Complete bed rest for seven weeks was ordered During this entire period the patient s condition was excellent The chest pain did not reappear and the only drug used was phenobarbital which helped on several occasions to allay restlessness and combat insomnia

At the end of seven weeks a gradual increase in activity was prescribed The anginal pain did not reappear Ten weeks after the attack an ortho diagram showed no cardiac enlargement Three months after the attack the patient was allowed to undertake light duties around the firehouse Since there was no return of either chest pain or dyspnea during the next three months a still further increase in his activity was permitted

This patient was examined at regular intervals from 19э4 to 1940 He has remained free of all symptoms The heart has not increased in size The electrocardiogram has returned to normal (see Fig 236)

The feature of considerable interest in reviewing this case is the complete disappearance of anginal seizures following an attack of acute coronary thrombosis A tempting explanation is to suppose that the branch of the coronary tree that was responsible for the angina was the seat of more advanced sclerotic changes Occlusion followed by healing that the complete bed rest permitted eliminated this myocardial sector as a focus for pain production The subsequent history of this patient suggests that the remaining coronary arteries if sclerosed are certainly not narrowed to a point where ordinary exertion produces symptoms Due credit must also be allowed for the collateral circulation that took place about this area

The fact that few drugs were employed in the management of this patient deserves comment An initial dose of morphine was given during convalescence an occasional dose of phenobarbital was used and when he returned to work a prescription was written for 2 grain capsules of theophylline ethylene diamine to be taken after meals Nitroglycerine was prescribed and carried at all times but since angina did not reappear it was never used After taking the capsules containing the xanthine derivative for a few weeks the patient was advised to go along without drugs No symptoms reappeared consequently no medication has been necessary during the past few years

A follow up study is now made every three months At each visit an interval history is obtained the heart size checked by roentgen examination and electrocardiographic examination repeated

This patient s record points out that the prognosis in coronary occlusion in some cases is excellent Younger patients tend to do very well if other factors in the history are good The favorable circumstances here include the absence of hypertension and cardiac enlargement and the fact that the anginal seizures did not recur when activities were resumed In addition, the attack itself was readily controlled, recovery prompt pain not prolonged, the fever was not high following the accident and congestive failure and abnormal rhythms did not complicate the picture An unfavorable factor that cannot be overlooked in this case is the poor family history

Many physicians, no doubt, could collect a number of similar instances of recovery following attacks of acute coronary occlusion in which the proper period of bed rest has been obtained and the return to ordinary activities has been gradual Rest undoubtedly is the main feature of the treatment

### Coronary Disease Complicated by Angina and Thrombosis—Improvement Following Careful Regulation

**Case 41** C S an electrical engineer of 53 was first seen in September 1935 complaining of chest pain on exertion for one year In June 1935, an attack of very severe chest pain was experienced lasting three hours and requiring a hypodermic of morphine for relief Bed rest for six weeks was followed by recurrence of anginal pain on slight exertion The patient returned to work two months following the attack but found that activity was considerably curtailed by chest pain

Physical Examination B P 114/70 The heart was enlarged to the left and a soft systolic murmur was heard over the mitral area The heart sounds were normal and the rhythm was regular

The orthodiagram (Fig 112A) showed a slight generalized enlargement The cardiothoracic ratio was 0 57 The transverse diameter was 14 3 cm (predicted 12 8 cm) cardiac area 13 sq cm (predicted 110 sq cm) aorta 3 8 cm

The electrocardiogram (Fig 112B) suggested that the recent severe attack of pain was caused by a coronary occlusion The infarct was thought to involve the anterior part of the left ventricle and the septum

The blood Wassermann was negative The blood count kidney function and concentration tests were all normal

Clinical Diagnosis A Etiologic Arteriosclerosis B Anatomic Cardiac hypertrophy Relative mitral insufficiency C Physiologic Anginal syndrome Normal sinus rhythm D Functional Classification Class 3 Therapeutic Classification Class C

Discussion A history of anginal attacks of a year's duration followed by an occlusion and persisting chest pain is not a very encouraging combination The amount of structural damage evident on physical examination that was confirmed by the electrocardiogram and orthodiagram made me hesitate to give this man s family anything but a poor prognosis However let us see what he accomplished

The patient s history revealed that he lived in the suburbs in a three story house on a hill Trips up the hill especially in winter weather were always attended by a great deal of precordial pain His work was difficult and he was constantly under a severe nervous strain In addition there was considerable difference of opinion between the patient and the manager of his department These emotional upsets increased the frequency

of the anginal attacks during the day There was no time allowed for lunch on many working days and consequently a very heavy dinner was the rule in the evening Excess tobacco was consumed at all times cigarettes during the day and cigars at night Later it was brought to light that the patient was active in a country club and held a minor township position both of

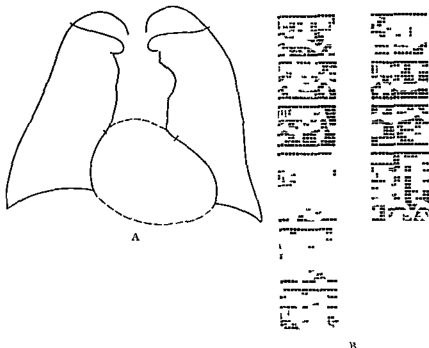



FIG 11? A Orthod a ram Note slight card c enlargement and hypertensive shape Ascend ng ao ta prom nent on r ght border The aortic knob promin nt B Electro card ogram of 6/8/35 shows nverted T I with do n ng of the S T interval in the same lead The direct leads (old technic) show bsence of Q aves n leads 4 and 5 and upr ght T waves n same lead The tracing taken on 1 /15/39 shows less marked change Lead 4F ho ever s suggestive Note sharply inverted T ave and the doming of the S T interval

which consumed the greater portion of his spare time in the evenings and on week ends

The first step toward readjustment of this man s activities was taken when he moved out of the house on the hill and secured a first floor apartment in the vicinity of the suburban railroad station This eliminated hill climbing in the evening Meanwhile the patient s wife learned to drive a small car and acted as chauffeur on all occasions In fair weather she drove the patient to the door of his office building and in bad weather to the railroad station An hour was taken for a light lunch When oppor tunity presented the patient was transferred to another department in hi

company that gave him a slight reduction in salary but shorter hours, a new department head and eliminated trips on the road

The anginal attacks gradually became less frequent and less severe Instead of 12 to 14 nitroglycerine tablets a day, he found that he needed only two or three Lighter meals were taken at night, and tobacco and coffee were eliminated A longer night's rest was at first made possible by a sedative but later this was withdrawn As soon as possible the patient resigned from his township office and the country club and replaced his golf by developing an interest in book collecting This was a fortunate hobby to choose and many of his evenings and week ends were spent in reading and discussing his books When summer came a two-months vacation was arranged and a bungalow rented at the seashore, where the same program was continued

This patient has now had five years of comparative comfort Anginal attacks have gradually decreased, although when he was last examined (December 15 1939) he reported an average of one or two slight seizures a day He has learned to take a nitroglycerine tablet under the tongue just before undertaking exertion that previously brought him a little above the pain threshold In this way he is increasing his collateral circulation for when the dilator drug opens up the coronaries who can say that the light exercise that follows does not force blood along new pathways?

The interest taken by this patient in getting well and the great help given him by his wife in every detail of his program account for the good result I doubt if any surgical operation directed toward increasing the collateral flow could have given any greater relief than was obtained on the medical regime The last electrocardiogram (see Fig 112B) taken five years after the first still shows evidence of the old infarction in the pronounced inversion of the T-waves in lead 1 The heart size decreased during this period (see Fig 112A)

This case history again illustrates that drugs play a very minor role in the treatment of coronary lesions A carefully planned program given to a patient who has the will to survive often produces a similar result If one *of the newer coronary dilator drugs had been given to this man* continuously during his course of treatment much undeserved praise would have been given to an agent acting only in the role of a placebo

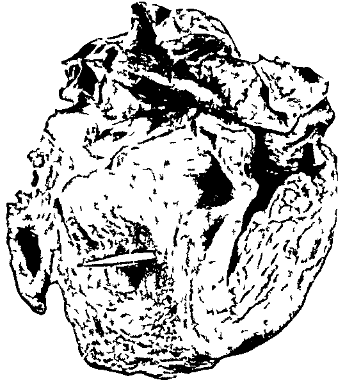
This patient also demonstrates the need for individual treatment in coronary disease The plan of battle should be drawn up when the full account of each hour of the day is at hand Faith should then not be pinned on a multiplicity of drugs (that are usually changed or rearranged at each office visit), but on the result of the correction of many problems brought to light by a complete understanding of the patient s temperament, occupation and home environment

## Coronary Occlusion (Unsuspected)—Sudden Death Following Rupture of Left Ventricle Through Area of Infarction

Case 42 W L a white male of 80 wandered into the Philadelphia General Hospital on *11/7/33 He was confused and disoriented* but showed a fair state of nutrition



A



B

Fig 113 Cardiac rupture through area of infarction on the posterior surface of the l ft entri le A S te of rupture b h nd posterior papillary muscle B Posterior a p ct Th re was an occlusion of the right coronary art ry ab ut 2 cm from its orifi e The aorta showed advanced sclerot c changes (Autopsy No 8236 Philadelphia General Hospital )

PHYSICAL EXAMINATION The physical examination was normal except for the changes in the eyes and cardiovascular system that are commonly met in senescence There was no cardiac enlargement no arrhythmias were present and no murmurs were heard The heart sounds were weak and distant

LABORATORY DATA The Wassermann was negative the blood count normal and the urine showed only a slight trace of albumin The blood urea was 19 mg per cent

During the following two weeks the patient showed little change in his mental or physical condition On the morning of the 17th hospital day he was found dead in bed

AUTOPSY (Fig 113) The pericardial sac was found to contain ?00 cc of bloody fluid and a large blood clot A small mottled rupture was seen on the posterior surface of the left ventricle Heart weight 440 Gms Coronaries prominent tortuous and sclerosed Myocardium mottled and flabby The area toward the apex resembled scar tissue of a healed infarct On opening the heart the site of the rupture was seen behind the posterior papillary muscle There was a small break in the endocardial surface about 5 mm in diameter No clot was attached The mitral and aortic valves were slightly thickened Both coronary orifices were markedly sclerosed especially the left About cm from its orifice the right coronary artery was found to be occluded It contained an adherent dark red clot extending into the two branches for several cm The aorta showed advanced sclerotic changes

DISCUSSION (Dr Martin P Crane*) Of all the types of cardiac involvement discussed so far the one most likely to result in sudden death particularly in a patient of this age is disease of the coronary arteries In the Philadelphia city morgue where more than 2 000 cases of sudden unexplained and violent death are examined annually this group constitutes the largest percentage Coronary occlusion due to atheromatous change in the vessel wall with subsequent narrowing and reduction of the blood supply is the most common finding [33] The formation of a thrombus in such a vessel occurs in a substantial percentage of cases but is by no means discovered as often as diagnosed Embolism involving the coronary mouths is a rare occurrence in our experience although for some unexplained reason this is frequently the diagnosis made by physicians summoned to pronounce death I have seen only one patient in six years in whom such a diagnosis was justifiable This patient a man of 40 years of age had been operated upon for the removal of a large pilonidal cyst, and at autopsy an embolus was demonstrated blocking the mouth of the left coronary artery and protruding slightly into the aorta The coronary vessels otherwise were in excellent condition throughout The diagnosis here incidentally was not made ante mortem

Autopsies performed to investigate the cause of sudden death in cases in the age group of the patient whose history appears above generally show the late results of coronary disease i e single or multiple areas of infarction aneurysmal dilatations of the ventricle or rupture of the myocardium through an area of softening in an infarct The most remarkable feature of many of the individuals presenting extensive lesions is the paucity of symptoms they appear to have exhibited before death according to the histories we obtain from their relatives or associates

Another observation that has interested us is the absolute lack of corre

* Chief Coroner s physician City of Philadelphia

lation between the extent of the infarcted or scarred area and death Many hearts have revealed such massive and obviously long standing lesions that we have wondered many times how the patient was able to survive and carry on strenuous activities in spite of such extensive myocardial damage Many of the infarcts that are encountered are often larger than a silver dollar On the other hand in some cases where death takes place suddenly no infarction may be present or if it is found after a search it may not exceed a ten cent piece in size In these cases we must seek the cause of death in the profound disturbances of cardiac rhythm initiated by this small area in the myocardium

In syphilis of the aorta simple narrowing of the coronary orifices without occlusion may produce sudden death (page 210) In such hearts the condition of the coronary vessels may be quite satisfactory beyond their point of origin in the diseased aorta

Hypertensive cardiovascular disease is in our experience second to coronary disease as a cause of sudden death It is of course frequently associated with coronary artery disease although it may operate as an independent factor in the production of the terminal episode Hypertension and syphilitic aortitis particularly in negroes appear to be a common combination in the production of sudden cardiac failure The enlargement of the left ventricle and the sudden demand for increase in the coronary blood flow in the presence of such a marked narrowing of the mouths of the coronary vessels in the aorta are the factors that explain the terminal episode

The effect of a sudden mental shock in these individuals was well illustrated by two cases observed recently at the city morgue A male negro collapsed and died suddenly while at work on a laboring project His body was brought to the morgue his associates meanwhile going to his home to carry the news to his wife She promptly collapsed and was pronounced dead in a few minutes Her body was likewise removed to the morgue Their hearts were identical in revealing syphilitic aortitis and the effect of long continued hypertension with marked left ventricular hypertrophy They differed only in weight the heart of the husband weighing 650 Gm, and that of the wife 600 Gm

Syphilitic aortic regurgitation and arteriosclerotic aortic stenosis are the chief valvular lesions that are associated with sudden death Rheumatic valvular disease is a distinctly rare cause in our autopsy experience at the morgue While it may occur it is less frequent than either of the two *previously mentioned* The youth of the patients who develop rheumatic lesions the comparatively little myocardial damage some of them reveal after the active process subsides as well as the wide clinical recognition such valvular lesions enjoy probably account for the fact that we see them so infrequently in our department Congenital lesions such as pulmonary stenosis and its combinations are in my experience rarely the cause of sudden death

Rupture of an aortic aneurysm syphilitic in origin is by far the most

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* Chief Coroner s physician City of Philad lphia

A suitable hypertensive regime was prescribed (page 305) and the patient was given instructions in adjusting his work to the level of his myocardial reserve

When next examined 14 months later all the signs of acute coronary occlusion were present Morphine was given at once for pain Three injections of 15 mg (¼ grain) each were required at two-hour intervals For the dyspnea and cyanosis oxygen was administered through a nasal catheter (page 99) at the rate of five liters per hour It was continued for 36 hours Three injections of 50 cc of 50 per cent glucose were given during the first 24 hours at eight hour intervals The patient showed gradual improvement following these measures and on the seventh day normal rhythm returned spontaneously (see Fig 243C) No digitalis was prescribed The remaining seven weeks of his period of bed rest were uneventful

When slight exertion was allowed at the end of this time a marked increase in the amount of dyspnea was observed His activities were restricted in an attempt to conform to this reduction in myocardial reserve and he was kept on one floor of his home for another month At the beginning of the fourth month the patient insisted on returning to his work as janitor of an office building Although he was advised against undertaking the work incident to this position the patient managed to go along for a year during which time he continuously showed signs of mild congestive failure He was digitalized two months after his attack of coronary thrombosis Intermittent claudication appeared during the first few months following his return to work although anginal pains were never experienced The claudication no doubt was more efficient in limiting his activity than the advice we gave him In addition to the maintenance digitalis tablet of 0 1 Gm (1½ grains) daily theobromine sodium acetate 0 3 Gm (5 grains) was given after meals No improvement in the claudication was noted

Two years after the acute occlusion edema of the feet became marked and persisted The heart showed increase in size and the exercise tolerance showed a further reduction Ordinary activity produced marked dyspnea Weekly injections of mercupurin (2 0 cc ) were given with ammonium chloride 1 0 Gm (15 grains) after meal this regime removed all evidences of edema

Two months later however the patient began to have frequent attacks of nocturnal paroxysmal dyspnea and died suddenly during one of the seizures No autopsy was obtained

Following the posterior coronary occlusion which must have been a large one this patient did not have enough cardiac reserve to carry on without discomfort He was always on the verge of congestive failure Dyspnea was marked on slight exertion and later dyspnea of the paroxysmal nocturnal variety entered the picture Death occurred during one of these sudden episodes of left ventricular failure Reviewing this record it is remarkable

how long the patient was able to survive following first, the acute failure of his coronary circulation, and later, cardiac failure of the congestive type

The most interesting feature of his single attack of coronary thrombosis is the sudden onset of heart block When this occurs the blood supply to the bundle of His has usually been sharply curtailed and momentary cessation of ventricular activity cerebral anoxemia and Adams Stokes seizures are not uncommon sequelae It is strange that this patient never lost consciousness when his pulse fell suddenly to 40 As a rule the greater the degree of bradycardia, the more serious the situation becomes and the more likelihood there is that Adams Stokes seizures will develop The sudden appearance of bradycardia of this degree following the attack of chest pain enables the clinician to make a diagnosis of posterior occlusion and to predict that the lesion is in the right coronary artery The infarct that results usually includes the posterior portion of the interventricular septum and the posterior surface of the left ventricle Since the infarct faces posteriorly, no friction rub is heard

It will be seen (see Fig 243C) that normal rhythm returned later This may be interpreted as evidence of the development of a collateral circulation in the tissues about the bundle On the other hand adequate anastomoses in the septum may prevent the occurrence of heart block when occlusion involves this region

In some instances following an acute coronary occlusion intraventricular or bundle branch block may accompany an A V conduction defect in which event the gravity of the situation is increased [13] [99] While complete heart block complicates coronary occlusion in from 0 7 to 4 0 per cent of the cases,[19] [6] intraventricular block has a much higher incidence Master has reported this finding in 15 per cent of 375 cases As a group these patients are older and present evidence of more advanced cardiac damage

Usually the complete block is permanent (page 406) in fact this arrhythmia may be the only remaining evidence of a previous occlusion when the patient is seen some time later Many of the unexplained prolongations of the P R intervals that are often encountered later in life no doubt represent a more gradual interference with the coronary blood flow Repeated occlusions have a tendency to increase this conduction defect until permanent complete block occurs (see Fig 181)

### Acute Coronary Occlusion Complicated by Complete Heart Block—Death During an Adams Stokes Seizure

**Case 44** W S a railroad engineer of 69 was first seen in April 1934 complaining of vertigo and dyspnea

Physical Examination B P 170/100 There was cardiac enlargement chiefly left ventricular aortic dilatation and an advanced degree of cerebral sclerosis

Laboratory Data The urine showed fixation of the specific gravity The blood Wassermann and blood count were negative

Cour f On these findings the patient obtained a railroad pension and with increased rest and digitalization showed few circulatory symptoms for a period of five years During this time he was examined three times and the ortho lagram and electrocardiogram showed no significant changes

In August 1939 there was a sudden attack of chest pain accompanied by sweating and signs of shock His course was rapidly down h ll in spite of the usual measures (morphine oxygen and glucose) Twelve hours following the onset of chest pain the pulse suddenly dropped to 45 and dur ng the next six hours he had frequent Adams Stokes seizures in one of w h ch he died (see Fig 44)

**Discussion** Complete heart block following the sudden coronary occlu sion in this patient was accompanied by Adams Stokes seizures When these attacks complicate coronary occlusion the outcome is usually fatal The treatment should be directed toward increasing the pulse rate and may be planned accurately only when the cardiac mechanism during the seizure has been determined by an electrocardiogram Epinephrine is safe to use in cases of ventricular standstill but in the presence of coronary disease it should always be used with care Management of Adams Stokes seizures is discussed in detail on page 403

MANAGEMENT OF THE CARDIAC COMPLICATIONS OF DIABETES MELLITUS

In the era before insulin coma acidosis infection and malnutrition were the chief causes of concern Hospital admissions in coma were not uncom mon Today diabetics do not die in coma and in fact are rarely seen in this state They are living longer and in increasing numbers are showing advanced changes in the coronary arteries [177] The chief cause of death in diabetic patients is now arteriosclerotic heart disease Symptoms of coronary disease are much more frequently met in diabetic patients than symptoms arising from arteriosclerotic involvement of the vessels of the brain periph eral arteries or other sections of the body

Many theories have been advanced to explain the close association between arteriosclerosis and diabetes Joslin[176] believes that the increase in blood cholesterol bears a direct relationship to the arterial changes evi dent in both young and old diabetic patients The frequency of the onset of diabetes in later life has suggested to some that the disease is the result of arteriosclerotic involvement of the pancreas which is only a part of the widespread process However postmortem studies that show the high incidence of arteriosclerosis in the young diabetic patient are certainly not in agreement with this view Angina pectoris and coronary occlusion are more common in women with diabetes than in nondiabetic women of the same age group which is further proof of the role of diabetes in the production of these vital changes

There is a greater incidence of arteriosclerosis in poorly treated or neglected cases of diabetes The longer the time before the disease is recog nized or properly managed by diet and insulin the greater the degree of arterial change that will be evident on clinical examination Nathanson [77] has shown that a high degree of vascular disease may be found in the young diabetic who survives for a period of ten years or more In an analysis of 100 autopsies upon diabetics this observer found an incidence of 41 per cent of severe coronary disease Above the age of 50 years the incidence was found to be 51 7 per cent as compared with eight per cent in an even larger series of nondiabetics of the same age

Hypertension is very common in diabetic patients Bell and Clawson [6] state that the incidence of hypertension is five times as great among diabetics as among nondiabetics This in turn has an influence on the degree of arteriosclerotic change in the coronary tree The increase in the blood pressure so common among diabetics cannot be attributed to the arteriosclerosis Obesity is certainly a factor that deserves consideration, as well as the variety of metabolic alterations that accompany diabetes

Many theories have been advanced to explain the development of arteriosclerosis (page 541) Since the majority of cases of diabetes in our country are over 40 an age when sclerotic changes are frequently encountered in nondiabetic patients the problem presented is by no means an easy one Furthermore there are no differences in the fundamental character of the *pathologic change in the two groups of patients Often a high degree of* sclerosis can be demonstrated in young people who have diabetes that has been improperly controlled To explain this, we again come to cholesterol, a substance that no doubt holds the key to the mystery The faulty metabolism that produces high concentration of this substance in the blood may have a direct relationship to the sclerosis The high blood sugar of the diabetic is likewise not a silent chemical abnormality

The effect of the dietary beliefs that characterized the diabetic management during different epochs may provide some important clues in the future in regard to the etiology of arteriosclerosis In the days before insulin the high fat low carbohydrate schedules favored sclerosis High carbohydrate diets combined with proper insulin regulation may be shown in the future to slow the process

Explanations of some of the other aspects of the problem of arteriosclerosis and its association with diabetes are not available For example why the same pathologic change appears to affect one section of the arterial system to a greater degree than another section remains unsolved

Since the important cardiac lesion in diabetes is coronary sclerosis it is not surprising that the incidence of angina pectoris in diabetic patients is high Root and Graybiel[3] [1] reported 210 cases of angina from a series of 7 000 cases of diabetes Of these patients 122 were males and 88 females The much higher relative proportion of females in this series emphasizes the importance of diabetes in the etiology Angina may occur in the diabetic patient who has had the disease for some years and it is more common in patients who have had indifferent treatment Consequently when angina appears in young individuals, in addition to searching for evidences of syphilitic infection it is extremely important to think of the possibility of diabetes

The presence of diabetes usually increases the gravity of the prognosis in angina In a series of 136 fatal cases reported by Root and Graybiel the average duration of life from the time of the first attack of angina was two years the majority of deaths (52 5 per cent) occurring during the first year The early detection of diabetes followed by careful treatment is therefore extremely important in the prevention of angina

In some cases marked coronary sclerosis may be present and give no symptoms until occlusion occurs If this possibility is overlooked and attention focused on the diabetes a diagnosis of diabetic coma might possibly be made However an estimation of the blood sugar and an electrocardiogram usually make the differential diagnosis

In the management of the cardiac patient who has well established coronary disease it is extremely important to realize that the diabetes may be much improved by accurate measures whereas the symptoms of coronary disease may grow proportionately worse I have seen serious accidents result from a too rapid reduction in the blood sugar level Over dosage with insulin with the sudden production of hypoglycemia is usually responsible but an extremely low carbohydrate diet may have the same effect In these cases prompt relief may be obtained by supplying the heart muscle with the necessary glucose by the use of intravenous injections of 20 cc of a 50 per cent solution

In the treatment of coronary disease in an elderly diabetic patient, insulin is not contraindicated It is well to begin with a diet slightly under the total energy requirements protein 2/3 Gm per kilo carbohydrate 3 Gm per kilo and fat to make up 15 calories per kilo Insulin should be started in very small doses and increased with caution until all sugar disappears from the urine Frequent blood sugar determinations should be made in order that reactions may be avoided A protective dose of 15 Gm of carbohydrate in the form of orange juice two hours after insulin is a great aid in preventing hypoglycemia in the treatment of elderly patients with coronary disease Fear of insulin shock however should not prevent the careful therapeutic use of insulin in this group Overdosage will give untoward effects but this is equally true with regard to many other valuable remedies that we use in daily practice Insulin carelessly given will cause cardiac pain which may be dangerous Insulin cautiously given to the same patient with a properly calculated diet will give great relief

In rare instances of spontaneous hypoglycemia (hyperinsulinism) the predominating symptom in older patients may be chest pain[38] Here the relationship of angina to hunger should serve to arouse the suspicions of the examiner Relief of the attack following the ingestion of sugar in a readily assimilated form is prompt I have seen one patient of this type a woman of 52 who was first treated by a neurologist for ' petit mal At the onset weakness frequent mental lapses and anxiety were prominent symptoms On questioning this patient when she was sent to me because of the additional symptom of precordial pain she stated that one of the nervous habits that she was instructed to overcome was excessive appetite To hide her increasing desire for food she made frequent visits to a neighborhood restaurant when she felt a spell coming on She even provided for emergencies when the restaurant was closed by hiding pound boxes of candy at strategic points about the house The first time I examined the patient I prescribed a reduction diet and nitroglycerine These directions

were quickly changed when further study, in addition to the signs of moderate sclerosis and slight cardiac enlargement, showed a fasting blood sugar of 60 mg per cent

The management of attacks of coronary thrombosis in diabetic patients with the exception of careful dietary regulation and insulin differs in no respect from the regime outlined on page 272 The insulin will usually be *found to be insufficient in most cases and will have to be carefully increased* If a few days elapse before this higher blood sugar level is discovered no harm results while hypoglycemia produced by careless insulin dosage in these acute cases may be dangerous

Congestive failure while not a common complication in diabetic patients usually causes a rise in the blood sugar and additional insulin must be administered as indicated Treatment of the edema consists in the use of digitalis (page 76) and diuretic drugs A low carbohydrate diet is useful *and small frequent feedings should be given (Chapter 21)*

### Diabetes Mellitus Complicated by Arteriosclerosis with Angina and Intermittent Claudication

**Case 45** M F a Jewish merchant of 44 was first seen November 7 1938 complaining of precordial pain and shortness of breath

History In the fall of 1936 diabetes was first discovered and the patient was placed on a diet with insulin On exertion at this time he complained of pain in the calves of both legs which was relieved by rest Six weeks before the first examination he noticed *tightness in his chest on exertion this was also relieved by rest* An occasional spell of dyspnea was experienced during the night Recently he had a severe seizure that required a hypodermic of morphine for relief The patient s father had hypertension His mother had diabetes

Physical Examination Height 5 5" weight 200 pounds B P 106/60 Pulse 80 Marked pallor slight dyspnea Heart sounds weak and distant Rhythm regular Slight cardiac enlargement to the left The liver was not palpable Marked sclerosis was evident in peripheral vessels No edema

Laboratory Data Orthodiagram (Fig 114A) shows cardiac enlargement of the hypertensive type The electrocardiogram (Fig 114B) shows T wave changes in all leads the most marked alterations appearing in lead 4F

Diagnosis A Etiologic Diabetes Arteriosclerosis B Anatomic Cardiac enlargement Old coronary infarction Coronary sclerosis C Physiologic Anginal syndrome Paroxysmal cardiac dyspnea D Functional Classification Class 3 Therapeutic Classification Class E

Discussion The family history here is most significant Both the patient s mother and father were overweight In addition his mother had diabetes and his father had hypertension

This patient at 44 showed evidence of advanced arteriosclerosis in eye grounds and in peripheral vessels The cramp like pains in the calves of the legs on walking that were relieved by rest and the characteristic chest pain confirmed the impression of widespread arteriosclerosis

The obesity must be considered an important factor in precipitating the diabetes which may have been present and unrecognized for many years prior to the time he developed intermittent claudication and first visited his physician

The attacks of dyspnea at night point to left ventricular failure secondary to the coronary disease Theophylline ethylene diamine was given in doses

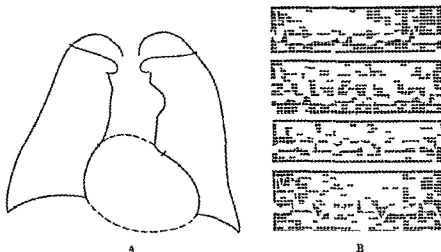



*Fig 114* A *The orthodiagram Cardiac enlargement is present B The electrocardiogram* Marked T wave changes are present in all leads The most characteristic deformity is seen in lead 4F Here there is slight elevation of the RS T interval above the isoelectric line and the T wave is deeply inverted There is a small Q wave in lead 3

of 0 3 Gm (2 grains) after meals and hypodermic injections of morphine for severe nocturnal dyspnea Following the last attack of dyspnea the patient was digitalized

# 9

# HYPERTENSIVE CARDIOVASCULAR DISEASE

Diseases never follow the same pattern even those which are placed in one group There are no more two diseases identical in course than there are two leaves of a plant exactly similar
—CORVISART

*Essential hypertension and its complications are problems of everyday* practice Since the hypertensive process once established places a great strain on the circulation many of the complications that arise are related to the cardiovascular system In some patients the duration from onset to heart failure is a short one, others show a great tolerance to the presence of an increased blood pressure over long periods and cardiac symptoms occur late if at all in the course of the disease

The terms employed in the past to designate this type of cardiac disease have been responsible for much confusion Of these chronic myocarditis is one still used by many physicians to designate any type of nonsyphilitic heart disease seen after the age of 40 Although this simplifies their diagnostic problems considerably the use of the term does not give full recognition to the fundamental causes of the heart failure in many of the cases Furthermore the term myocarditis aside from being a bugbear to statisticians is in itself misleading There is no inflammatory process present in the hearts of this group even when examined microscopically If the presence of fibrous tissue is discovered in the myocardium it is by no means proof of a pre existing inflammation since it may be the result of antecedent coronary occlusion with infarction of a large or small area of heart muscle Cardiorenal disease is also an unsatisfactory term to use in referring to cases of cardiac enlargement accompanied by signs of cardiac (rarely renal) failure

Hypertension is merely a symptom However when it becomes established *and there is continued elevation of both systolic and diastolic levels* of the blood pressure we speak of the condition in the absence of a known cause as essential hypertension This condition we are apt to regard as a disease entity While it is true that its cause or causes are still unknown, recent investigations are clearing the picture and many of the widely scattered facts in literature may be utilized in treatment Consequently an inquiry into the known causes of hypertension seems appropriate at this time

## ETIOLOGY

The classification of Williams and Harrison[400] on page 294 is an excellent etiologic summary

This grouping shows that a variety of clinical conditions may cause elevation of the systolic and diastolic blood pressures

**Among the neurogenic** are included first of all the psychoneurotic group often mistaken clinically for thyrotoxicosis in whom marked fluctuations in blood pressure occur following emotional upsets Other symptoms of neurosis are present and for this reason sedatives like phenobarbital are a great aid in management In other cases following long periods of stress and strain we may observe an increased blood pressure The correction of environmental influence in these instances tends to restore the blood pressure to normal

**The injuries to the brain stem** in the region of the vasomotor center which occur in diphtheria poliomyelitis and encephalitis and are accompanied by high blood pressure levels are included under the medullary form The hypertension in these cases disappears following recovery The association of hypertension with the increased intracranial pressure of skull fractures is well known

Laboratory animals often show increase in blood pressure following reflex stimulation of the carotid sinus and aortic depressor nerves In man elevation of the blood pressure during an attack of angina or following a coronary occlusion may occasionally be the result of a reflex mechanism from the heart Often this elevation will disappear following recovery from an attack of occlusion

**Endocrine Factors** When the action of the various endocrine substances on the circulation and blood pressure became known it was not unusual that the attention of many investigators should turn in this direction for a solution to the riddle of hypertension The suprarenal medulla was investigated and found to secrete a very powerful hormone possessing a constricting action Proof was thought conclusive when hyperplasia of this area was found in some hypertensive individuals and when patients with adenomas of the suprarenal cortex exhibited hypertension However evidence to the contrary soon accumulated for all hypertensive patients did not show change in the region of the suprarenal cortex The theory that hypertension is caused by excess adrenalin secretion has been further exploded by the failure of modern delicate methods to detect adrenalin in amounts in excess of normal in the blood of hypertensive subjects

Pituitary disease may be associated at times with hypertension when there is excessive secretion from the posterior lobe (basophilic adenoma) Here again adequate proof is lacking since attempts to demonstrate excess of this hormone in the circulating blood of hypertensive patients have failed

It is a common observation that vasomotor disturbances occasionally associated with increase in the blood pressure accompany the menopause

TABLE VII

CLASSIFICATION OF HYPERTENSION*

(R pr te l by perm on of the a th and th d t of th An l of Intern l Med )

I NEUROGENIC

A Psychogenic
  1 Psychoneurotic §
  2 Stress and strain
B Medullary
  1 Diphtheria §
  2 Poliomyelitis §
  3 Encephalitis §
C Increased intracranial pressure §
  1 Carotid sinus †
  2 Aortic depressor nerves †
  3 Ischemic muscle
    a Cardiac
    b Skeletal

II ENDOCRINE

A Pituitary (basophilic hyperplasia—Cushing s syndrome)
B Adrenal
  1 Medullary adrenalin (pheochromocytoma) †
  2 Cortical tumors
C Ovarian
  1 Menopause
  2 Arrhenoblastoma

III RENAL

1 Acute and chronic glomerular nephritis §
2 Obstruction to urine flow §
  (a) Congenital anomalies §
  (b) Ureteral stricture §
  (c) Urethral obstruction
  (d) Pelvic tumors
  (e) Spinal bladder
3 Urinary tract infection
  (a) Pyelitis §
  (b) Pyelonephritis (classical or masked) §
4 Diseases of renal arteries
  (a) Renal atheroma (large and small arteries)
  (b) Arteriolar sclerosis ‡
  (c) Infarcts of kidney
5 Tumors of kidney
  (a) Wilms tumor §
  (b) Other tumors
6 Coarctation of aorta
7 Renal calculi

IV METABOLIC

1 Hypercholesterolemia (renal atheroma ?)
2 Gout (uric acid deposits in kidneys ?)

V CONGESTIVE HEART FAILURE

VI MIXED AND UNCLASSIFIED CAUSES OF HYPERTENSION

* Th s does t l d pat t w th l t f y t l p ly
§ These cond t a l k l ses f l ated blood p es h l f y g f lt
† D m t ated a m l b t t i m
‡ Alth ugh rteriol scl os p b bly i t t d by hyp t m t t tt l t c se f th se blood p e

However only a small percentage of women develop hypertension at this time and certainly not a greater number than the hereditary tendency to this disease would account for The disturbed relationship between the endocrine glands or the endocrine balance as we like to call it because of the presence of less ovarian secretion at the menopause may explain the pressure increase in these cases The same hypothesis of imbalance may be utilized to account for the association between hyper and hypothyroidism diabetes and hypertension

**Renal Factors** Hypertension may be renal in origin This thought was originally advanced by Richard Bright when he suggested a causal relationship between renal disease and cardiac enlargement Recently the investigations of Goldblatt and his colleagues have again focused attention on the kidney as a cause of essential hypertension These workers began with the premise that hypertension is associated with vascular disease of the kidney The renal circulatory changes they believe are associated with ischemia By using an adjustable clamp they produced constriction of any desired degree of the main renal artery of laboratory animals In 1932 Goldblatt and his associates reported the production of persistent hypertension in dogs for the first time by renal ischemia These results were soon confirmed and studies to determine the mechanism of the production of the hypertension followed Although space is not available to discuss these important contributions in detail [4] the possible mechanisms whereby hypertension can result from renal ischemia will be mentioned

Goldblatt first considers the increase in peripheral vascular resistance that comes about in order to elevate the pressure and improve the blood flow through the ischemic kidney This may be produced by a nervous reflex from the kidney that is effective through the vasomotor apparatus It may also be induced by the formation of a substance by the ischemic kidney which accumulates in the blood and acts directly on the contractile elements of the arteries or indirectly by first affecting the nervous vasomotor apparatus Such a substance however has not been demonstrated

Nervous Reflex All the experiments of Goldblatt and his co workers that have been performed to date tend to eliminate a nervous reflex from the kidney as the mechanism responsible for increased peripheral resistance On the other hand their published studies point to the likelihood of a humoral mechanism of renal origin as a cause for the increased peripheral vascular resistance that produces the elevation of the blood pressure Whether the substance is secreted or excreted by the ischemic renal parenchyma and how it finds its way into the circulation are unsolved problems

Endocrine Stimulation Increase in blood pressure may also be caused by stimulation of the endocrine glands by the same unknown substance from the ischemic kidney The hypophysis and the adrenal glands have been studied with this thought in mind but the experiments have been inconclusive

\ 1 1 1 1 3 1?4 1 5 1 6 1 7 1 8 1 9 130 131 13

RENAL HUMORAL MECHANISM The present status of his studie on experimental hypertension has been summarized by Goldblatt[1 4]

> it appears to be established beyond reasonable doubt that the hypertension which develops after constriction of the main renal arteries or as a result of renal ischemia produced by any method is due to some humoral mechanism of renal origin Evidence is accumulating to justify the conclusion that the results of these studies on animals may be directly applicable to the pathogenesis of both the benign and malignant phases of essential hypertension in man which is associated with the presence of intrarenal or extrarenal vascular or other disease that can produce renal ischemia Further knowledge of the pathogenesis and perhaps treatment of this condition will depend upon the establishment of this conclusion

COARCTATION OF THE AORTA is included under the renal heading in the classification proposed by Williams and Harrison since experimental narrowing of the aorta above the level of the renal arteries is followed by hypertension while constriction below the renal arteries will not produce it The renal origin of many cases of hypertension has undoubtedly been overlooked until recent years The management of these patients now includes a careful renal study, and high percentages of abnormal pyelograms have already been reported

**The metabolic group** includes patient who have gout with an increase in the blood uric acid, also those with hypercholesterolemia Even in these gouty individuals urate deposits in the kidneys have been considered in relation to the occurrence of hypertension

**In heart failure** the systolic and diastolic pressures may fall to normal levels or below and improve when the congestive manifestations fade from the picture However in other cases failure may cause hypertension to appear and improvement is accompanied by a fall in pressure These unusual cases are given a separate grouping in the classification

***Mixed or Unclassified*** *Finally it is obvious that in many instances* more than one factor may be operative in the production of the hypertension while in others no cause whatsoever will be evident after an exhaustive study has been carried out These cases are grouped under the heading of mixed and unclassified causes

## MALIGNANT HYPERTENSION

When the appearance of essential hypertension is followed by rapid changes in all the arterioles of the body we speak of the condition as malignant hypertension This is not a separate disease but merely a variety of essential hypertension that appears in a different pattern It is a leaf of the same plant growing a little more rapidly to wither and die ahead of the others

In malignant hypertension the factor or factors concerned in the hyper-

tensive process operate in such a manner as to speed up the sequence of events The pressures particularly the diastolic seek higher levels at which they are sustained, intracranial pressure rises and the afferent glomerular vessels become constricted The latter change occurs quickly leading to an irreversible narrowing in the vessel wall

### Incidence

**Race** The incidence of hypertension appears to be increasing a fact that we are in the habit of blaming on the tempo of modern existence We know that hypertension is rare among the Chinese and other races in the Far East who lead more tranquil lives and among African negroes in the primitive state On the other hand hypertension is quite common among negroes living in large cities in America occurring more frequently and at an earlier age in them than in members of the white race Diet and manner of living are the factors that are most often invoked to explain this difference

**Age and Sex** Life we like to be told begins at 40 If this is so perhaps it is more than coincidental that hypertension selects the same period of life for its onset in over 80 per cent of the cases Cardiac complications appear about ten years later Hypertension is equally common in both sexes The lighter occupations undertaken by women may explain the greater mortality rate observed among men and the quicker course the disease takes in the male once it is established

**Familial Tendency** Every general practitioner knows that angina and cerebral hemorrhage appear more often in some families than in others and that either may be the final result of a long standing hypertensive process (Fig 115) A characteristic bodily configuration is generally observed Hypertensive or sthenic types of patients are inclined to be obese and have broad deep chests and short thick necks Obesity hypertension and diabetes are closely allied disorders that appear too often in certain families to be explained on any other than hereditary grounds According to the statistics furnished by different observers from 50 to 70 per cent of all patients who have essential hypertension give a family history of the condition

Susceptibility Is there any way of sorting out in the early years those patients who will eventually show a permanent increase in the blood pressure level? This is obviously an important question and closely allied to *treatment for application of protective measures* at an early age might reduce the incidence and the complications in the later years In a person predisposed to hypertension we have reason to suspect that the defect lies in part at least in the sympathetic system Environmental stimuli produce changes in the blood pressure more readily in these individuals than in normal persons of the same age Although these variations are at first intermittent if the stimuli that produce them are continuously repeated the changes may become permanent

TEST FOR SUSCEPTIBILITY A very simple test has been suggested to determine the presence of this over reaction of the blood pressure to stimu

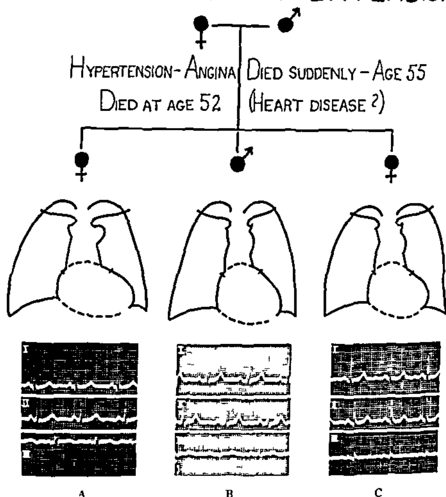



FIG 115 A Housewife of 48 Dyspnea and anginal pain present for two years Overweight B P 210/120 Sclerosis of retinal vessels Marked cardiac enlargement Systol murmurs over apex and aortic areas Electrocardiogram (above) shows marked left axis deviation B Mechanic of 45 Slight dyspnea on exertion Overweight B P 180/110 Slight sclerosis of retinal arteries Moderate cardiac enlargement Soft systolic apical murmur Electrocardiogram (above) shows a slight left axis deviation C Housewife of 42 Dyspnea on exertion for two years No other symptoms Overweight Moderate cardiac enlargement No murmurs Electrocardiogram (above) normal B P 170/100

lation If the patient's hand and arm are placed in ice water at 4° to 5 C (39 2° to 41° F ), a strong stimulus is produced and the blood pressure

rises In normal patients the elevation in systolic and diastolic pressure will amount to about 10 mm of mercury In patients who are predisposed to hypertension the blood pressure will rise from 30 to 70 mm of mercury This often accounts for the high blood pressure seen when this group of patients is examined for the first time by an insurance company physician In a brief period their pressures return to normal and the finding is usually thought to be of no consequence However if these patients are carefully studied subsequently by the physician the family history of hypertension will be found to be positive in well over 80 per cent of the cases For this reason the careful recording of blood pressure values by life insurance examiners industrial and school physicians in patients who would not ordinarily come under the physician s care becomes a matter of great importance Here early changes may be noted at a time when effective preventive measures may be planned

Various poisons have long been mentioned as leading factors in the development of hypertension Lead has been known to exert a vasoconstricting effect on the arteries but it is doubtful if this effect can be a continued one and account for a chronic hypertensive process Tobacco may have some effect on angina and peripheral artery sclerosis but it has not been shown to produce a chronic increase in the blood pressure Alcohol likewise cannot be said to play a significant role in the increase in heart disease at least not through the production of hypertension

Finally about all we can say in regard to the cause of essential hypertension today is that it follows a widespread vasoconstriction of the arterioles of the body cause unknown which increases the work of the heart leading to hypertrophy and eventually to failure A more rapid progression of these changes we refer to as malignant hypertension

## SIGNS

When the chronic vasoconstriction of hypertension becomes established clinically detectable changes in the heart and arteries are not long delayed The late Richard Cabot summed up the situation to his classes briefly The findings in hypertensive heart disease consist of high blood pressure and a big heart Hypertrophy of the left ventricle is first to occur since it is this chamber that feels the strain of the abnormally high peripheral resistance If vasoconstriction continues and increases dilatation of the left side of the heart occurs with widening of the mitral ring and the systolic murmur of relative mitral insufficiency is heard in the region of the apex

Increase in the pulmonary blood pressure now follows As the right ventricle receives the strain, it responds in similar fashion by an increase in its size When it can no longer carry the burden it in turn shows dilatation and signs of failure While these changes are taking place in the heart and lungs the aorta is receiving an increased amount of trauma at each systole If viewed under the fluoroscope a widened arch may be noted although this change is not as marked as that observed when

syphilis is present The elevated blood pressure is now reflected in the increase in the pitch of the aortic second sound As the disease advances, degenerative changes appear in the aorta, small tears may occur, caused either by the direct impact of the blood column or by the twisting effect it exerts on the whole arch Later, if these areas give way before the increased force of the blood column dissecting aneurysms may form

Long-continued strain on the peripheral arteries produces a thickening or sclerosis The coronary arteries are so located that they cannot avoid the damaging effects of the column of blood delivered to them under such an increased pressure and all too often rapidly progressive changes in these vessels lead to an early fatal issue particularly if the picture is further complicated by the presence of diabetes

## SYMPTOMS

The patient who has a well established hypertension may have no symptoms whatsoever that would lead him to consult a physician and many times the elevation of the blood pressure is first brought to light by a life insurance examination The course of the disease from this time depends a great deal on the skill of the physician first consulted in explaining the nature of the condition to the patient and on the type of nervous system possessed by the patient who receives this information In some patients a host of symptoms of psychoneurotic nature can be traced back to the day they first learned of the increase in blood pressure To them this figure represents a definite disease and their initial fright prompts them to travel from physician to physician seeking a magic remedy for their only complaint 'blood pressure In other patients, from the very onset the hypertensive process may pursue a swift malignant course with wide spread changes and many symptoms that are real, progressive and uncontrollable

**Dyspnea** Between these two extremes there are a number of patients who show structural alterations of a slowly progressing type The first symptoms to appear are usually of a cardiac nature of these increasing dyspnea on exertion is the most common Observing this symptom as the hypertensive process advances it will be seen to grow gradually worse and even if the patient is overweight (which is usually the case) the dyspnea may be in excess of the amount noted on the previous examination Dyspnea is the earliest sign of weakness of the left ventricle and if uncontrolled by appropriate measures will progress gradually or rapidly to orthopnea and other evidences of congestive cardiac failure

If failure of the left ventricle occurs quite rapidly in patients with an established hypertension a sudden attack of dyspnea may occur This is known as acute paroxysmal dyspnea or cardiac asthma It comes on frequently at night when during sleep the patient slips into an unfavorable position Some cardiac patients may be comfortable in one recumbent position and uncomfortable in another There is a quick awakening with

an increase in the respiratory rate and a sense of suffocation The spell may disappear with change in position or it may become aggravated by the increased respiratory movements which favor a greater venous return to an already overloaded heart Pulmonary congestion increases producing further reflex respiratory stimulation [143] The congestion that follows edema of the lungs places an additional burden on the circulation

Pulmonary edema may come on rapidly during an attack of cardiac asthma Rales appear and these become more numerous and widespread as the spell progresses A frothy blood tinged expectoration often follows an attack in some cases

Cardiac asthma is a common symptom in patients who have had high blood pressure for some time with continued overstrain of the left ventricle The sudden depletion of cardiac reserve following a coronary occlusion may precipitate an episode of this paroxysmal type of dyspnea Cardiac asthma may also complicate the course of chronic nephritis less often it may be secondary to an energetic right ventricle in mitral stenosis suddenly pumping more blood into the lungs than is able to pass the narrowed mitral valve (see Case 15) Too great a volume of fluid intravenously * abdominal distention overactivity cough constipation fright and nightmares as well as faulty posture during sleep are other factors usually blamed for precipitating attacks of paroxysmal dyspnea The relationship between allergy and hypertension is discussed in Chapter 16

**Heart Symptoms** While the continued elevation of the blood pressure causes cardiac failure in most cases intercurrent infections may add the deciding burden that precipitates cardiac breakdown Often when the cause of failure is not apparent clinically an extensive coronary involvement may be discovered at postmortem In some cases anginal pain may have suggested the complication of coronary sclerosis while in others the first intimation of the presence of coronary disease may be a sudden coronary occlusion

Disturbances of cardiac rhythm frequently account for the palpitation complained of in many cases of hypertension Premature beats are common When there is a persisting total irregularity auricular fibrillation is the usual cause Paroxysms of tachycardia or flutter are rare but may occur Advancing coronary disease interfering with the circulation to the bundle may produce varying degrees of heart block

**Nervous symptoms** may precede or accompany these cardiac manifestations and are often so pronounced that they tend to obscure the picture Vertigo headache insomnia tinnitus and inability to concentrate are common complaints of patients who have established hypertension Nose bleeds occur in some instances and may precede a cerebral hemorrhage In hypertension the peripheral circulation may be curtailed to the extent that symptoms are produced in the form of disturbances of sensation (parathesis) or pain on walking (claudication)

* Warning against over-energetic postoperative treatment

***Visual symptoms*** (headaches blurring of vision amaurosis) may appear early in the course of the disease, and the patient will first consult

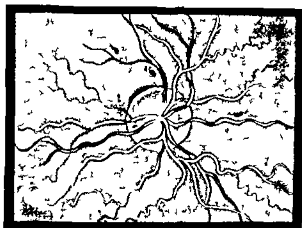


Fig 116 Retinal changes in essential hypertension

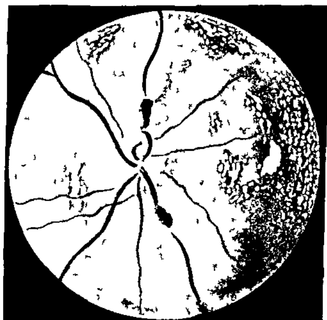


Fig 117 Retinal changes in malignant hypertension

an ophthalmologist at which time the presence of essential hypertension will be revealed by an examination of the visual fields (Fig 116) Experience in the use of the ophthalmoscope is essential in the treatment of hypertension since changes in the eye grounds are common and the

information gained by this examination is useful in diagnosis as well as in prognosis A narrowing of the arteries of the retina which may be the result of constriction or sclerosis is first seen If the change is marked it usually means an advancing lesion Many times the blood pressure of a patient may be normal when examined in which event the retinal arteriosclerosis may be the only evidence on physical examination of a previously existing hypertension

Malignant hypertension also leaves its stamp on the eye grounds in the form of papilledema, retinal lesions and advanced narrowing of the vessels (silver wire arteries) (Fig 117) These changes may be observed far in advance of the renal alterations that characterize the terminal stages of the disease

## DIAGNOSIS

**Cardiac Hypertrophy** It is necessary to demonstrate the presence of cardiac hypertrophy before a diagnosis of hypertensive heart disease can be made This increase in heart size even if the blood pressure happens to be normal at the time should always suggest hypertension in the absence of other causes for enlargement If closely questioned on these occasions the patient will usually give a history of a previous elevation of the blood pressure and often a later reading after circulatory improvement has taken place will show a higher level

**Murmurs and Sounds** In hypertension systolic murmurs are usually heard over the cardiac apex and in the aortic area while the second heart sound in the aortic area is accentuated Rarely when there is a marked degree of aortic dilatation a functional diastolic murmur may be heard in this region I have seen only two cases of functional aortic regurgitation secondary to hypertension Both came to autopsy and syphilitic aortitis was proved to be absent in each instance When left ventricular failure develops during the course of hypertension and the pulmonary circulation becomes engorged the pulmonic second sound may increase until it equals or exceeds the aortic second sound in intensity

If auricular fibrillation is absent a gallop rhythm may also be heard accompanying the failing heart in hypertension (page 15) The third sound that makes up the gallop usually follows the second sound of the heart and is therefore protodiastolic It should be distinguished from the normal third heart sound when the latter is present This is easy as a rule since third heart sounds occur in normal subjects with slow heart rates When a gallop rhythm is heard in patients with hypertension a block of one of the bundle branches should always be suspected The asynchronous contraction of the ventricles that occurs in the presence of this lesion causes splitting of the first heart sound usually best heard between the apex beat and the left sternal border

**Pulsus Alternans** When congestive failure complicates hypertensive

heart disease another sign that should be searched for is pulsus alternans (page 407) for if present, this arrhythmia aids in establishing prognosis Appearance of either pulsus alternans or gallop rhythm in the early stages of failure in hypertensive heart disease is usually indicative of a poor outlook

Other organs in the body that are involved in the hypertensive process may furnish clues that may help to identify the type of cardiac damage if the blood pressure happens to be low when the patient is first examined Kidney lesions may be suspected in the presence of albumin, a low fixed specific gravity casts and nitrogen retention, although these signs if they appear first with the onset of congestive failure, should not be viewed as conclusive evidence of renal damage Transient paralyses, aphasias, as well as headaches point to cerebral changes that may be the result of either angiospasms or cerebral hemorrhages

Hypertension may be said to be present when the systolic blood pressure is persistently above 140 mm of mercury or the diastolic above 90 mm of mercury However many variations occur When first seen the blood pressure in most hypertensive subjects is already established at a higher level the average reading being 170/100 An occasional patient will show a systolic reading as high as 300 mm of mercury Many retain the same blood pressure level for years and feel much worse when attempts are made to lower it In the presence of heart failure or following a coronary occlusion the blood pressure may drop to a normal level or lower The cause of the cardiac hypertrophy is then obscured and the examiner must turn to the history and the physical examination for further evidence Often the diastolic level will not drop to the same extent and will remain well over 100 mm of mercury This is a valuable sign On the whole too much stress is placed on the blood pressure figure itself at the expense of the more significant features of the physical examination

The characteristic alterations in the orthodiagram in hypertension are discussed in Chapter 1 (page 44), and the electrocardiographic changes in Chapter 24

A careful physical examination including the usual laboratory tests will in most cases furnish all the data necessary for a diagnosis of hypertensive heart disease The presence of cardiac enlargement without valvular disease establishes the diagnosis at once in the presence of elevation of the blood pressure If the pressure is not elevated careful attention should be given to the history in order to exclude patients with coronary disease who frequently exhibit cardiac enlargement In these cases the eye grounds may contain the evidence necessary in making the differentiation If the history is atypical and the eye grounds negative it may be most difficult if not impossible to make the distinction without further observation In some cases when congestive failure is successfully treated by appropriate therapy the blood pressure may return to its previous high level, and this observation will often clear the diagnosis

## PROGNOSIS

Accurate prognosis is most essential in this type of heart disease and although there are wide variations a few general rules may be stated Usually the older the patient the better the prognosis since in younger patients the disease is more apt to run a malignant course Patients who first develop hypertension in the fifties or sixties may show few untoward effects

The blood pressure figure itself usually has little significance However if the diastolic pressure remains above 120 there is usually trouble ahead The disease in these cases may take an abrupt malignant course with a quick termination In any event when a diastolic pressure of 120 mm or over is discovered at each examination the duration of life rarely exceeds two years

**Better Prognosis in Women** Women in my experience are able to withstand the effects of a high blood pressure exceedingly well for many years Mild elevations of the systemic pressure that are seen at the time of the menopause may persist and cause little difficulty in later life The course of hypertension in women is less apt to be complicated by coronary accidents

**Complications** Aside from the development of malignant hypertension with its rapidly fatal course complications may at times arise in any case and determine the outcome Congestive cardiac failure is the most common and terminates the disease in over half of the cases Cerebral hemorrhages coronary disease and a progressing kidney lesion in the order named are the next most common terminal events in hypertension Complications therefore arise mainly from the heart and the brain and not as formerly believed from the kidney

The cardiac involvement secondary to hypertension is usually progressive and as a rule the larger the heart the poorer the prognosis Gallop rhythm and pulsus alternans also point to a poor prognosis When attacks of cardiac asthma complicate the picture the duration of life is rarely over a year The presence of angina makes prognosis difficult The patient is always liable to sudden death but with careful management may live a number of years

The assessment of the renal lesion aids in prognosis and in most cases a concentration test will give a good index of renal function If there is fixation of the specific gravity in a young person with hypertension the outlook is grave Older patients may show slower progression of the kidney lesion When uremic symptoms are added to the picture at any age the prognosis is most serious

## TREATMENT

In planning the management of any case of hypertensive heart disease due consideration should always be given to the underlying cause or

causes if they are evident after a thorough physical examination Treatment directed toward the renal endocrine metabolic or neurogenic factors (page 294) whenever possible may lighten the cardiac load and postpone the onset of circulatory symptoms

## Daily Regimen

If attention is focused on the patient as a whole and his problems studied in relationship to his environment a therapeutic program may be worked out for each case The object of present-day therapy is not to lower the manometer reading to a figure considered normal for the age of the patient We do not possess any drug or method that we can depend upon to accomplish this permanently The aim in treatment should be to guide the patient in the adoption of a sane mode of living in keeping with his clinical condition This includes suitable exercise, proper dietary instructions, the prevention of complications and the relief of any annoying symptoms that develop in the course of the disease We should also try at all times to steer a course away from the many fads of the day If the information is requested the patient should be told the blood pressure level for nothing is to be gained by keeping the matter a secret in a day when patients drift from one physician to another and athletic clubs department stores, and even the boardwalk at seaside resorts have their blood pressure stations

If there is no cardiac enlargement, particularly in women at the menopause the physician may be justifiably optimistic Likewise in cases of well established hypertension where the patient has progressed well from year to year the records of previous examinations speak for themselves The patient of this type should be told how innocuous an increase in the blood pressure can be in many instances Proof can be furnished that it is a condition compatible with old age A great deal of time must be given to explanations following the first complete physical examination, particularly if there are no symptoms present and the physician does not prescribe any medicine

The patient who has hypertension should be educated to meet all situations in his business or in his home with calmness and equanimity and to avoid conflicts as much as possible During recent years few patients could be advised to seek new jobs Instead they have had to recondition the old ones to fit a changing physical state Much the same advice can be given to both coronary and hypertensive patients in regard to exercise recreation vacations and other matters of the daily regime

If the patient cooperates in these fundamental matters he may in a very short time feel rested and improved in which event his blood pressure may seek lower levels without the benefit of drugs If drugs had been administered they would have been given credit for in this manner false therapeutic reputations are built by a variety of substances from carbon

dioxide to watermelon seeds when enthusiasm lifts them to the realm of cures

Ayman[7] has collected over 200 accounts of procedures and drugs that have been proposed for the treatment of high blood pressure A detailed review of 35 of these showed that all laid claim to either partial or complete success It is evident that the encouragement received with the remedy and the confidence the patient has in the physician go far in bringing the degree of relaxation necessary for symptomatic relief In the early stages of some of the neurogenic types this improvement may be long continued

Buck points out the usefulness of treating cases of hypertension by the methods that have proved their value in the management of the psychoneuroses and recommends similar class instruction This psychologic approach may be useful as a method in large city hospitals for providing the necessary information in regard to diet exercise relaxation and mode of living to large groups who would otherwise receive no instruction in these necessary fundamentals

## Drugs

**Nitrites** Of the various drugs that have been recommended for essential hypertension the nitrites appear to be the first thought of many practitioners Consequently a few words regarding their place in the therapeutic program is in order It is not good therapy to administer one of the nitrite group to be taken at stated intervals as soon as hypertension is diagnosed While it is quite true that the nitrites under some circumstances are excellent in reducing arterial pressure their action is fleeting and frequently if given to patients with advanced arteriosclerosis they produce symptoms that are always annoying and at times may be actually dangerous A sudden lowering of the blood pressure is likely to cause syncope in older people Little is gained because the blood pressure rises again in a short while following the use of the dru and remains elevated until the next dose is taken In the absence of symptoms what benefit could the patient possibly derive from these short periods of blood pressure decline? The use of the nitrite group should therefore be confined to patients with angina pectoris and those showing vasospastic crises and should always be governed by the need of the moment

Nitrites produce vasodilatation because of their direct action on the smooth muscle of the vessel walls They depress systolic pressure to a greater degree than the diastolic and this suggests that their action on the smaller arterioles is less marked A description of the various nitrite preparations in common use will be found on page 247

**Bismuth subnitrate** has recently enjoyed a wide reputation that is chiefly based upon the belief that it is slowly absorbed from the gastrointestinal tract where it supplies a small quantity of nitrite gradually thus sustaining its effect and overcoming this objection to nitrite therapy In

the doses suggested by Stieglitz,[3, 6] this remedy has not been successful in my hands

**The iodides** have been advised in small doses in the treatment of essential hypertension and have continued to hold their place through the years Some claim a vasodilating effect follows their use others like their alterative action" The majority of physicians who use the iodides in hypertension find them just as good and not as dangerous as the hundreds of newer remedies whose main action is to depress the blood pressure level When advocates of iodide therapy are overenthusiastic in their claims I always suspect that the hypertension in the patient who shows such striking results is either complicated by syphilis or is secondary to thyrotoxicosis

**Sedatives** are most useful in the treatment of some types of hypertension and have gained a reputation by their action on the accompanying *nervous symptoms They are invaluable in irritable and excitable patients* in securing the rest and relaxation so necessary in successful management In small doses 0 15 to 0 30 Gm (¼ to ½ grain) every four hours phenobarbital will exert a sedative effect rather than a hypnotic one and its administration may be accompanied by a decrease in the blood pressure level Bromides may be continued to advantage combined with the elixir of phenobarbital in some cases Chloral hydrate is a safe and effective sedative and in the usual dosage 1 0 Gm (15 grains) has no untoward effect on the heart

**Endocrine products** have been advocated for the hypertension that accompanies the menopause[7] Here again the effect may not be a direct one since the fall of blood pressure may follow the relief of the annoying symptoms that accompany the condition Other glandular extracts (liver pancreas or thyroid) have as far my experience goes, no definite place in the management of hypertension

**Other Drugs** The remaining host of preparations described in the *numerous pamphlets that increase the bulk of the daily mail* have as far as I know not been proved to be of exceptional value in the management of essential hypertension I mention in passing the sulfocyanates (this group may be actually dangerous) cucurbocitrin benzyl benzoate adenosine adenylic acid acetyl choline and histamine

## Other Measures

**Physiotherapeutic methods** *in the treatment* of essential hypertension appear to have their greatest value when combined with suitable rest which explains the good reports that follow when they are used as a part of the regular regime of spa treatment Diathermy and carbon dioxide baths are also popular adjuvants at the various resorts (Chapter 19)

**Routine venesection** is not to be encouraged as a beneficial therapeutic measure in hypertension since the blood pressure very quickly returns to its original level when the blood volume is restored However when heart failure comes on with increase in the venous pressure prompt venesection of 500 cc is one of the most efficient methods of restoring cardiac balance

The above measures directed toward relief of the symptoms that commonly arise in the course of essential hypertension constitute the treatment of the patient who has hypertensive heart disease which is asymptomatic If no evidence of congestive failure exists in the absence of auricular fibrillation digitalis should not be given When this drug is needed the presence of hypertension is no contraindication to its use Many times there will be a rise in the blood pressure following the clinical improvement that accompanies digitalization but this should never be regarded as a matter of any consequence On the contrary it shows the ability of the myocardium to restore the blood pressure to the customary level where as the patient has demonstrated greater efficiency may be attained The subjective improvement observed in the patient generally confirms this opinion

**The increasing dyspnea of cardiac failure** calls for prompt and complete digitalization When congestive failure develops in hypertension the measures are the same as those used when failure complicates other types of heart disease These are fully discussed in Chapter 2

**Paroxysmal dyspnea or cardiac asthma** calls for special mention Prevention of the attacks is important and may be possible if attention is paid to the factors mentioned on page 74 A suitable bed (See Fig 38) should be procured whenever possible so that the patient may not run the risk of slipping down into the unfavorable position that initiates a seizure Sedatives should be prescribed to allay nervousness and to secure a full night s rest After recovery from the initial attack the patient should be digitalized This will be found to control or greatly diminish the severity of the seizures for a time in some instances although with increasing weakness of the left ventricle the attacks recur In many of these patients the use of diuretics will again secure some temporary measure of relief Theophylline is useful while the administration of one of the organic mercurial group at regular weekly intervals may be helpful in preventing attacks even in the absence of demonstrable edema (See Case 5) Renal function should be determined before this course of treatment is begun and the existence of a possible obstruction to the urinary flow ruled out In some cases the use of mercurial diuretics in preventing attacks of cardiac asthma in the absence of visible edema may be much more helpful than morphine particularly when vomiting uniformly follows the use of the latter drug Suitable limitation of fluid is necessary in these patients but with routine diuretics this need not be carried to the point where the patient is uncomfortable Care should be taken when diuretics such as mercupurin are used not to insist on too drastic salt restriction A slight readjustment may also be necessary in the daily maintenance dose of digitalis (page 113)

Angina pectoris and coronary occlusion not infrequently complicate the course of hypertensive heart disease Their treatment is discussed in Chapters 7 and 8

The increasing incidence of hypertension its complicating lesions in heart brain and kidney and the futility of most medical efforts to influence its course permanently once established challenge the efforts of all inves

tigators It is natural that the surgeon should become interested in this unsolved problem and not at all surprising that he should choose as his points of attack the endocrine glands and the nerves that carry the constrictor impulses The following brief summary of the surgeon's viewpoint in the treatment of hypertension is furnished by Dr James Lehman

## SURGICAL TREATMENT OF HYPERTENSION

Prior to 1925 the treatment of hypertension was entirely a medical problem Since that time there has been increasing effort on the part of numerous surgeons to perfect an operation for amelioration of the symptoms of high blood pressure These operations have been many and varied In attempts to solve the problem much needless surgery has been done and no doubt is still being done A number of theories have been advanced that attempt to explain the nature of hypertension but so far there has not been a single one that satisfactorily answers all the perplexing problems of management Consequently none of the operative procedure is entirely satisfactory in all cases While time has shown the fallacies of many of the earlier reports concerning operative relief in hypertension too much credence must not be placed in these suggestions no matter how clear and logical they seem It is an encouraging sign that during the past few years some agreement among surgeons has been developing as to the selection of the patient for operation and the type of operation to be performed.

Bitter criticism has been raised in some quarters against the use of surgery for high blood pressure which is after all only a symptom the cause of which is unknown While not denying any of the arguments against surgery much more valid proof has recently been obtained of the usefulness of surgery in arresting the progress of the disease and in producing an amelioration of the subjective symptoms On the other hand it must not be assumed that an operation will ever restore efficiency to an arteriosclerotic kidney nor elasticity to a hardened artery However if cases which are unresponsive to medical treatment are recognized early surgery may find a real place in the therapy of hypertension

No attempt will be made here to review the various operative procedures which have been employed in recent years by both American and European surgeons While there is still no unanimity of opinion the zone of action is narrowing to an area represented by the splanchnic nerves the celiac ganglia and the adrenal glands The operative measures may be classified under three headings (1) adrenalectomy (2) rhizotomy and (3) celiectomy

**Adrenalectomy** Acting on the theory that overactivity of the adrenals was the cause of hypertension many surgeons have practiced adrenalectomy The operation has been more popular in Europe than in this country Encouraged by the reports of cures following the removal of adrenal tumors surgeons wrongly interpreted this form of paroxysmal hypertension with the equally poorly understood essential hypertension Crile

(1916) was probably the first surgeon in this country to perform unilateral adrenalectomy for the relief of hypertension He soon discarded the operation after noting a return of symptoms at the end of one year De Courcy more recently advised bilateral subtotal adrenalectomy and attempted to establish an analogy between hyperthyroidism and hypertension He selects his cases early by tests of renal function and the fundus examination and performs his operations using the retroperitoneal approach through a kidney incision in two stages with an interval of two to three months[77] His statement that it is an accepted fact that hypertension is due to an increased adrenalin content of the blood is not in accord with present-day opinion My experience with this operation while small shows that the results are not permanent and the benefits if any are fleeting

**Rhizotomy** Adson and Heuer have been the chief proponents of rhizotomy They performed section of the anterior roots of the spinal nerves from the sixth thoracic to the second lumbar and found that this procedure was followed by an appreciable drop in pressure in all cases The pressure showed considerable variation with the position of the patient such a drop occurring in the erect position that abdominal support became necessary For this reason rhizotomy has been sharply criticized as a debilitating operation which seriously cripples the patient

Heuer used several criteria in the selection of patients for operation He chose cases where the blood pressure was not fixed and demonstrated this flexibility pre-operatively by making observations (1) after a period of bed rest (2) after the administration of drugs such as amyl nitrite and sodium thiocyanate (3) after the injection of colloidal sulfur or acetyl beta methyl choline The stage of the disease was determined in the usual way by renal function tests by ocular fundus examination and by a cardiac survey Heuer felt that the paralysis of the abdominal muscles was of little significance and was not particularly disabling The best results with this procedure were obtained in cases of malignant hypertension

**Celiectomy or Splanchnicectomy** Today the best results in the surgical treatment of hypertension follow one or both of these operations which are similar in theory as well as in technic The nerves may be severed either above or below the diaphragm Peet uses the intrathoracic approach resects a small portion of the eleventh rib on each side retracts the pleura and sections the splanchnic nerves The lower most portion of the gangliated sympathetic chain is also removed along with the rami communicantes from the tenth eleventh and twelfth intercostal nerves

Peet places great reliance upon the experimental work of Goldblatt and his associates and believes that in many of the cases he has operated upon hypertension is due to ischemia of the kidneys from overstimulation of the vasoconstrictors of the blood vessels that supply these organs He believes that his operation removes the nervous clamp from the kidneys and recommends the usual criteria in the selection of cases for surgery He also believes that a good result is possible in patients under 50 years

of age whose kidney function tests are still fairly good where ocular changes are not too far advanced, and in whom medical treatment has been tried with no appreciable improvement Some of his cases have shown amelioration of symptoms for as long as five years and most of his patients have obtained symptomatic relief

At the Mayo Clinic, the operation that is favored at the present time by Adson and his colleagues consists in bilateral section of the splanchnic nerves the upper two lumbar ganglia and the intervening sympathetic chain The subdiaphragmatic retroperitoneal approach is used

These workers are in accord with Peet in proposing the neurogenic origin of hypertension Overactivity of the vasoconstrictors they theorize is the cause of the symptoms, and their operation is performed to denervate completely the splanchnic vessels the kidneys and adrenal glands

In the careful selection of patients for operation many tests are per formed pre operatively Their observations show that many of the cases which seem best suited for operation give the poorest results postoperatively There are also certain sequelae of this operation caused by the removal of the lumbar ganglia i e loss of sweating in the lower extremities and increased skin temperature that should be noted The male usually becomes sterile however without the loss of libido There is no change in the menses of the female nor in the child bearing function

After a long period of experimentation Crile has evolved the operation of celiectomy Beginning many years ago with unilateral adrenalectomy he changed later to bilateral adrenal denervation while he now performs bilateral celiac ganglionectomy and denervation of the aortic plexus

Based upon comparative anatomic studies, Crile states that

> of highest significance is the role of the celiac ganglia and the celiac and the aortic plexuses which are related to the control of the speed of the circulation of the blood The control of the speed of the circulation of the blood includes the power to raise the blood pressure instantly It includes the quick control of the capillary bed control of the force of the heart beat In other words since the celiac ganglia the celiac and the aortic plexuses the splanchnic nerves and the sympathetic ganglia in the adrenal medulla are the exclusive mechanisms for normal adaptive con trol of the blood pressure and the blood circulation one would logically expect these to be the sole mechanisms which under pathologic activity would raise the blood pressure day and night for months and years until the kidneys the heart and blood vessels and the brain are injured that is are so changed that heart failure brain hemorrhage or kidney failure follow

He therefore concludes

> that celiac ganglionectomy and denervation of the aorta and of the adrenal gland could permanently affect only the physiologic

aspects of the disease and could not affect pathologic changes in the wall of the arterial tree and that in cases that have passed



Fig 118 Incision for celiac ganglionectomy

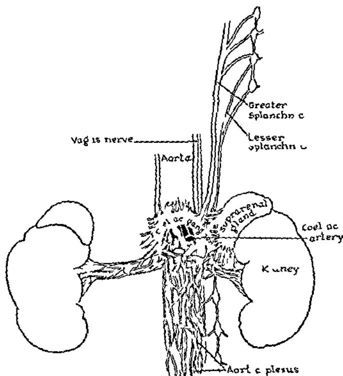



Fig 119 The left celiac ganglion and its connections

the limit of cure the progress of the disease may be slowed down or arrested Since a free plexus of nerves connects the celiac ganglia the adrenal medulla and the entire sympathetic net

work of the body there may be an overflow stimulation to the thyroid gland the pancreas the liver so that a group of symptoms—nervousness palpitation sweating, headache—are produced that is there results a general sympathetic drive These widespread and frequently distressing subjective symptoms are uniformly relieved by celiac ganglionectomy

Crile performs his operation under light gas anesthesia He uses the retroperitoneal approach through a lumbar incision (Fig 118) The celiac ganglion (Fig 119) is located by the sense of touch and dissection is carried out by the trained hand Visualization of the anatomy is not attempted In over 300 cases Crile reports some relief in 87 per cent and complete relief in 35 per cent one year after operation The diastolic pressure was reduced to normal in 39 per cent of the cases [7]

## ILLUSTRATIVE CASES

### Essential Hypertension of Long Duration in Spite of an Overabundance of Unnecessary Treatment

**Case 46** Mrs K K housewife was first examined in January 19 0 at the age of 68 There were no symptoms The blood pressure was 0/100 at that time During the following 0 years the systolic pressure varied from 180 to 30 and the diastolic from 90 to 100 An occasional headache one syncopal attack and vertigo all directly related to medication were the only complaints The exercise tolerance was never impaired and the mental status was excellent

The heart was not enlarged the rhythm always regular and the rate between 60 and 70 A systolic murmur was present over the entire precordium The electrocardiogram showed only a left axis deviation A slight trace of albumin was constantly present in the urine with an occasional hyaline cast Concentration tests showed a specific gravity of 1 016 to 1 0 Death occurred suddenly following cerebral hemorrhage at the age of 88 No autopsy

**Discussion** This patient a relative, was one of the first cases to come under my care She continued to carry out all my instructions and to take the multiplicity of remedies that were prescribed with a degree of faith that I have seldom seen equalled Reviewing my record of her case in the light of modern knowledge of the subject of hypertension, I marvel at her ability to weather trials of all the preparations administered in increasing numbers during these years

Going back 20 years to the time when all attention was focused on the manometer reading I first tried my skill at lowering this patient s blood pressure to a figure that I thought would be a more tolerable level for a woman of her age For the first therapeutic thrust I chose a potent weapon nitroglycerine A few minutes after she took the first triturate (1/100 grain) I received an emergency call to her side Vertigo and syncope had occurred and these were alarming symptoms in an elderly person who had had few complaints in the days before I attended her The blood pressure reading showed that there was no doubt about the fact that my remedy had the desired effect in lowering the blood pressure level How

ever in the presence of arteriosclerosis the sudden insufficiency of the cerebral circulation that resulted produced symptoms and as I was told symptoms had never been present before This was the last time that I prescribed nitroglycerine to be taken routinely by the patient whose only departure from the normal standards of health was revealed by a blood-pressure reading While the quickly acting members of the nitrite group are most valuable for the anginal seizures or the transient cerebral vascular spasms or crises that accompany hypertension they are harmful if given routinely in haphazard fashion

When this patient recovered from her syncopal attack and the first contact she had ever had with whiskey * I fell back on the iodides in order to secure more time to map out a different plan of attack on her blood pressure which (fortunately) at this point had completely regained its former level of efficiency

During the next few months although the blood pressure showed little change the patient was quite certain that she felt much better—at least much better than when she took the last medicine A few months later she was disturbed by many colds in the head and eruptions of the skin It looked like the end for the iodides but by this time elaborate plans had been formulated Sedative treatment could be given to allay the nervousness and lower the blood pressure consequently a sodium bromide phenobarbital mixture was prescribed after meals

A few months later the patient s family complained that Mamma is drowsy and queer A follow up visit at this time revealed an element of truth in both assertions The patient was in bed and examination showed lack of ability to concentrate a slow and indistinct speech and a desire to sleep at all times Although small doses of both sodium bromide and phenobarbital had been prescribed often in older people untoward symp toms may appear They are generally caused by the accumulation of the drugs in the body that follows faulty elimination by an arteriosclerotic kidney All symptoms promptly disappeared when the sedative mixture was discontinued

During the next few years this trusting patient accompanied me through the mistletoe days the benzyl benzoate outbreak the watermelon seed epoch and even cautiously sampled the thiocyanates While she had many more annoying symptoms than were formerly present was it not also true that the years were passing? She was now 80 years of age and should expect a few bad days now and then

A review of the findings at this advanced age showed a blood pressure that was still holding its former level of 220/100 no change in the heart size or the electrocardiogram and no symptoms other than those explain able on the basis of the remedies administered Profiting by this experience it was suggested to the patient that she continue without medicine at least for a time unless symptoms developed This was not a reasonable sug gestion to a patient of her age who had been raised on the belief that

* Formerly pop l r as first aid mea ure in all emergencies especially card c

medicine of some kind is indicated if any abnormality whatsoever is discovered by the physician 'For every disease there must be a *remedy*' Consequently my last therapeutic *sortie* in her case was a prescription for capsules of appropriate size containing milk sugar They were given with assurance and confidence and were accepted as representative of all the therapeutic knowledge of the day *in* the matter of hypertension * Immediate subjective improvement followed their use and the patient s statement that she had not felt so well in years I had no reason to doubt for in my own family circle at least I had come of age in the management of uncomplicated essential hypertension

## Hypertensive Cardiovascular Disease Complicated by Congestive Cardiac Failure—Autopsy

Case 47 P C a colored laborer of 42 was admitted to the Philadelphia General Hospital on 5/ 4/36 complaining of shortness of breath and cough

History For the past few years dyspnea had been increasing in severity Six months prior to admission severe attacks of dyspnea had been present at night These were occasionally followed by a blood tinged expectoration Edema was noted a month before admission increasing gradually No precordial pain had been present at any time Chancre in 1909 and antisyphilitic treatment in the out patient department from 1929 to 1936 No history of rheumatic fever

Physical Examination B P 140/105 Marked dyspnea and distention of the neck veins present *Heart* enlarged to the left as far as the anterior axillary line A musical systolic murmur was heard over the entire precordium with its greatest intensity at the apex A systolic murmur was heard over the aortic area The rhythm was interrupted by occasional premature contractions There were rales at both lung bases Shifting dulness was discovered in the abdomen and the liver edge was palpable below the right costal margin (Fig 120A)

Laboratory Data Urine (24 hour specimen) specific gravity 1 011 trace of albumin *Blood* count hemoglobin 70 per cent (Sahli) R B C 3 400 000; W B C 13 200 Kahn negative

*Roentgenogram* decompensating (rheumatic) heart disease with pulmonary congestion

*Electrocardiogram* (Fig 120B) Occasional premature contractions low voltage QRS flat T waves and left axis deviation (myocardial disease)

Course During the next two and one half months the patient continued to lose ground rapidly Gallop rhythm was present two weeks before death Attacks of paroxysmal nocturnal dyspnea increased in frequency and the patient died in one of the seizures on 8/7/36

Clinical Diagnosis A Etiologic Hypertension B Anatomic Cardiac hypertrophy Relative mitral insufficiency C Physiologic Paroxysmal cardiac dyspnea D Functional Classification Class 4 Therapeutic Classification Class F

Autopsy (Fig 120C) *Heart* weight 280 Gm Scattered throughout the left ventricle and auricular myocardium were numerous white scars about 4 mm in diameter with marked thinning of the myocardial wall at these points The valves were normal and the coronary ostia fully patent The aorta showed a few atheromatous plaque but beginning 4 cm above the aortic root were numerous small linear and stellate depressed intimal scar

Discussion The increase in the dyspnea over the course of a few years prior to death indicated a gradual diminution in the myocardial reserve Six months before admission the occurrence of attacks of paroxysmal nocturnal dyspnea gave evidence that the situation was becoming an acute one

* *As indeed they were considering her physical findings*

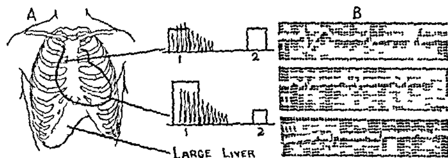



Fig 1 0 A Diagram of physical findings B The electrocardiogram Occasional premature beats of ventricular origin (first beat lead 1) The QRS voltage is low all leads The T waves are flat Left axis deviation C Hypertensive cardiovascular disease Cardiac hypertrophy and dilatation A few atheromatous plaques may be seen in the first part of the aorta (*Autopsy No* 31,914 Philadelphia General Hospital)

With the onset of congestion, the blood pressure dropped to 140/105. If the past history of the patient had not been known, some difficulty might have been encountered at this point in establishing the diagnosis of hypertensive cardiovascular disease. The age and the murmurs may have suggested rheumatic heart disease and the history of syphilis in a negro who showed evidence of heart failure may have caused luetic heart disease to be considered as the etiology. However there was no history of rheumatic infection, signs of mitral stenosis were absent and the rhythm was regular. We know that the patient had syphilis for which he received prolonged and satisfactory treatment. It seemed unlikely that heart failure could be caused by syphilis in the face of this and in the absence of aortic regurgitation to explain the large heart.

The most suggestive sign was the level of the diastolic pressure on admission even though the systolic level had dropped sufficiently to confuse the picture. The evidence of the previous existence of hypertension remained in the eye grounds and was reflected in the size of the heart. The murmurs could be explained also on the basis of relative mitral insufficiency following the increase in the size of the left ventricle and aortic dilatation secondary to the hypertensive process.

At autopsy the definite history of syphilis and the prolonged and adequate treatment that the patient had received over the course of some years led us to examine the first part of the aorta with care. There were only a few small healed scars visible in this region. These were considered to be unimportant clinically since they neither interfered with valvular function nor caused blockage of the coronary arteries. The value of antisyphilitic therapy in the prevention of the progress of aortitis is reflected in this instance (Fig 120C).

When this patient entered the hospital he was given a hypodermic injection of morphine and quickly digitalized. He received 0.2 Gm (3 grains) of theophylline ethylene diamine after meals. At the end of the first week thoracentesis on the right side yielded 900 cc of fluid. During one of the episodes of left ventricular failure that was attended by cyanosis and engorgement of the jugular veins a venesection was performed and 500 cc of blood removed.

## MALIGNANT HYPERTENSION IN A GIRL OF 21—FAILURE OF MEDICAL REGIME

**Case 48** Miss H M, a single American clerk of 21, was first examined in September 1934. The presence of hypertension had been noted a year before when she had a headache of three days' duration. Since that time headaches recurred so frequently that six months later she was forced to give up her job. Lately vomiting accompanied the headaches and she lost 15 pounds in weight. The headaches usually were present in the morning and grew worse during the day. For a month before examination she had noticed progressing weakness, vertigo, blurring of vision and palpitation. The patient's father died of a stroke at 50. Her mother at 40 had essential hypertension.

PHYSICAL EXAMINATION B P 40/130. Underweight. A slight exophthalmus was present. The skin was pale and dry. No thyroid enlargement. The eye grounds showed hyperemia and edema of both discs. Large hemorrhagic areas were present on both

retina a w ll a l r f th retinal art r s Th h art ho ed slight enlargem nt to th l ft an l th aortic on l un l s accentuate l

LABORATORY DATA Bl od count was normal th Wass rmann negative Blood urea 7 mg per 100 cc The urine sho ed a trace of albumin and the phenophthalein el mination n two hours was normal

CLINICAL DIAGNOSIS A Etiolog c Hypertension (mal gnant) B Anatomic Car l ac enlargement C Phys olog c Normal sinus rhyth n D Funct onal Classification Class I Therapeut c Classification Class D

COURSE Rapidly dow n h ll The headaches inc eased the renal lesion became apparent and the patient following a series of convulsions an l a hort period of coma died seven months later in the Philad lphia General Hospital

**Discussion** The diagnosis of essential hypertension usually presents no difficulty However the distinction between malignant hypertension and chronic nephritis usually requires a longer period of study

In this patient the absence of anemia the extremely high level of both systolic and diastolic pressure and the relatively good renal function present when first seen were all points favoring the diagnosis of malignant hyper tension The picture presented by the eye grounds was likewise suggestive There were no snow banks hyperemia of the disc was present in contrast to the anemia generally seen in nephritis and the retinal arteries reflected the changes that most commonly accompany hypertension

Younger patients are more apt to develop malignant hypertension Instances of this condition are reported in the literature before the age of puberty although this patient is the youngest that I have seen

The onset of the malignant phase of hypertension in this instance was typical Severe headaches usually call attention to the eyes and the dis covery of the advanced changes in retina and disc on ophthalmoscopic examination clinches the diagnosis

The extremely high systolic and diastolic pressures in these cases and the edema evident in the discs are signs that suggest the origin of the severe headaches Edema of the brain and increase in intracranial pressure are thought to follow the inability of the arterioles in this region to constrict to the same degree as is shown by similar vessels in other parts of the body The persisting elevation of the diastolic pressure likewise speeds the changes that usually are observed to follow a long continued hyper tension and the damage to the arterioles of the kidneys may be quickly reflected in the clinical picture Stupor convulsions and eventually uremia develop in a very short time

The medical management of the complications of malignant hyper tension is difficult and the relief obtained usually temporary The patient whose course is as rapid as the one presented here is always best treated in the hospital

Relief of the headache and vomiting is usually the first indication on admission When the blood count is normal the effect of venesection should be tried first In some cases this procedure may bring considerable initial relief of the headache When the symptom recurs the therapy should be directed toward the fundamental cause of the condition The edema of the

brain and increased intracranial pressure may be favorably influenced by intravenous injection of hypertonic sugar solutions Glucose (1200 cc of a 50 per cent solution) may be used but is apt to be followed by a secondary rise in pressure with recurrence of symptoms since it can diffuse into the brain and spinal canal Lately sucrose has been employed in the same concentration and amount and generally proves much more efficient sinc its administration is not attended by a secondary rise in intracranial pressure

Spinal puncture is a useful measure at times in the relief of the symptoms of hypertensive encephalopathy It should be done with care and judgment preferably in the hospital If the initial pressure is found to be high after the needle enters the spinal canal a slow withdrawal of sufficient cerebrospinal fluid is indicated to lower the pressure to normal Spinal tap however is attended by some degree of risk in these cases and the degree of relief it brings to the symptoms is by no means constant

Blackfan and Hamilton [9] have successfully used magnesium sulfate in treating the complications of vomiting and convulsions in these cases For the headache of malignant hypertension it is given by mouth or rectum (50 cc of a 50 per cent solution), or if convulsions develop, it can be administered intravenously (10 cc of a 2 per cent solution per kilo of body weight) The action of the magnesium sulfate when given by mouth or by rectum is brought about by dehydration The action when injected intravenously (or intramuscularly) depends upon the effect of the element magnesium upon the central nervous system Excitability is abolished because of paralysis of motor nerve endings

Sweating has no beneficial action on the cerebral symptoms of malignant hypertension and is no longer advised In some cases it may even be harmful

The nitrites are inefficient and powerless in combating a process of such severity as malignant hypertension, and usually show no appreciable effect on either the blood pressure or the symptoms

### Early Malignant Hypertension—Bilateral Celiac Ganglionectomy

**Case 49** R C a chauffeur of 9 when first examined in December 1938 complained of high blood pressure and nervousness

History Hypertension was discovered two years before first examination Considerable initial improvement followed a medical regime but during the following six months although able to work he felt very much worse Vertigo headaches biliousness and finally dyspnea and palpitation developed He lost ten pounds in weight

Physical Examination B P 240/130 Ophthalmoscopic examination revealed marked tortuosity and narrowing of retinal arterioles and a few scattered hemorrhages on the left retina Both optic discs were hazy The heart showed slight enlargement to the left There was a systolic murmur heard over the mitral area and the aortic second sound was accentuated

Laboratory Data Basal metabolic rate minus ten per cent Blood count normal Blood urea 38 mg per 100 cc Urine showed a light cloud of albumin and hyaline casts

Electrocardiogram (Fig 171A) showed inversion of T waves in lead 1

Clinical Diagnosis A Etiologic Hypertension (Malignant) B Anatomic Cardiac

enlargement Relative mitral insufficiency C Physiologic Normal sinus rhythm D Functional Classification Class I Therapeutic Classification Class C

COURSE Celiac ganglionectomies were performed on January 15 1939 and February 5 1939

DISCUSSION The problem presented by hypertension is indeed a grave one when we consider that 23 per cent of all deaths of persons over 50 are directly attributed to it * The internist has no specific therapy to offer in the management of this condition At the present time he meets the indi

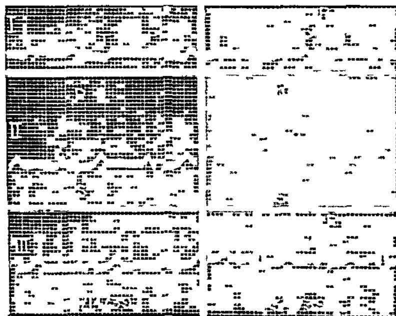


FIG 1 1 The electrocardiogram taken before operation (1/14/39) shows diphasic $T_1$ Following operation (3/11/39) the T wave in lead 1 returned to normal

cations as they arise and treats his patient by the use of carefully planned regimes the details of which have been previously outlined In many cases medical treatment meets all requirements However when the onset of a malignant course is evident surgical measures should be considered for in the presence of these rapidly progressing changes the medical man possesses no therapeutic weapon of value The surgeon on the other hand by his attack on the sympathetic nerve pathways reduces arterial tone at least in part of the vascular tree and in malignant hypertension has so far obtained results that are without parallel in medical treatment

Following operation the blood pressure of this patient was not restored to a normal level Before operation the average readings were 240/120 while during the first year after operation the average pressure was

* Four times the cancer death rate

160/100 However the subjective relief was marked and for this reason he was able to return to work He had few headaches less nervousness, and less exhaustion at the end of the day Furthermore, this improvement in his condition was reflected in the ocular fundi The spasm of vessels was less, and the retinitis disappeared The discs cleared The heart showed a decrease in size, and the T waves of the electrocardiogram became upright (Fig 121B) The albumin was less six months after operation and a decided improvement in renal function was noted

Before operation a marked response in the blood pressure followed immersion of the hand in ice water (page 298) After ganglionectomy this response was less marked but still in excess of normal

While we must admit that perfection in the treatment of hypertension must await further studies the relief that it is possible to bring to properly selected cases by the use of the surgical methods at our command today cannot be overlooked Haphazard surgery in poorly selected cases that have not first been given the full benefit of a medical regime of treatment tends to discredit all operative measures This is unfortunate when we consider that the surgeons have constructed their procedures along lines that conform to all the present-day concepts on the subject of hypertension

## DISSECTING ANEURYSM OF THE AORTA

A rare yet important lesion that may complicate hypertension is partial rupture of the wall of the aorta with dissection of its coats known as dissecting aneurysm Four times (that I am aware of) this accident has occurred in patients under my care In three of these cases that came to autopsy the diagnosis was missed while the fourth was diagnosed correctly If in any type of heart disease all possible happenings are constantly kept in mind there is less likelihood of a diagnostic error consequently it is worth while to review the outstanding clinical features of this unusual accident

Dissecting aneurysm occurs more frequently in males between 40 and 60 years of age who usually give a history of hypertension The first tear in the aorta is most often a transverse one just above the aortic valve through which the column of blood finds its way to the medial coat where it begins to dissect the vessel along the pathway of least resistance It may re enter the aorta at a lower point with the formation of a new channel of variable length (double barrel aorta) or it may extend down to the bifurcation and cause compression of the iliacs Half of the circumference of the aorta may be split to form this new channel and any arteries arising from the vessel along the course of the dissection may be involved Occasionally the dissection travels toward the aortic orifice in which event the coronary arteries are in danger of compression If perforation takes place through the adventitia sudden death follows with the escape of blood into the pericardial sac the pleural cavity or the abdomen

Dissecting aneurysm very rarely complicates syphilitic aortitis occasionally takes its origin at the site of an atheromatous ulcer but most frequently selects a spot where a previous medial degeneration has occurred The exact nature of this medial change that Erdheim has designated medionecrosis aortae idiopathica cystica is unknown Some claim that it is merely a degenerative process of old age while others state that it is infectious in origin

## SYMPTOMS

The symptoms attending dissecting aneurysm are sudden in onset and characteristic of the condition Pain is always present It is excruciating and persistent in spite of large doses of morphine Some patients describe it as a tearing pain Usually it is so severe and attended by so profound a degree of shock that consciousness is quickly lost The pain usually centers about the front or back of the chest but in some instances it may be felt in the abdomen in which event the condition is quite often mistaken for the surgical emergency of perforation or embolism (page 483)

The direction taken by the dissecting column often determines the symptoms (see Fig 157) Pressure on the coronary vessels may cause pain that cannot be distinguished from that of occlusion leakage into the mediastinum may produce hoarseness pressure on the renal arteries anuria while compression in the region of the iliac vessels gives rise to symptoms in the extremities Dyspnea is generally present A previous history of hypertension is nearly always obtainable in fact in spite of the shock and collapse the blood pressure may be maintained at a high level The pulse rate varies but is generally increased at the time of the rupture becoming more rapid until it is imperceptible in the presence of internal hemorrhage

Usually death is sudden The longest survival after the onset of pain in the cases I have observed was 48 hours Longer periods have been reported in a recent excellent summary of 19 cases of dissecting aneurysm by Glendy Castleman and White [117] One of their patients lived for 105 days after the formation of the dissecting aneurysm and died suddenly 12 hours after its rupture

The roentgen ray examination may suggest the diagnosis In one case (see Fig 32) the roentgen ray study made on different days revealed a shadow in the left chest following the accumulation of blood in the pleural cavity An increase in the prominence of the aortic knob may occur in other cases following the accident

The electrocardiogram is valuable in ruling out the diagnosis of coronary occlusion It will be negative unless blood flow through either coronary artery is blocked by the dissecting column of blood

Coronary occlusion is the usual diagnosis made in these patients because of the sudden onset the severe pain and (if the patient survives) the subsequent fever and leukocytosis Aside from the electrocardiogram the

points in the differential diagnosis favoring dissecting aneurysm are (1) the tendency of the blood pressure to remain elevated in the presence of so serious a catastrophe (2) the excruciating pain which reaches its peak at once (3) the more widespread radiation of the pain (4) the development of symptoms suggesting obstruction of the circulation at distant points and (5) the absence of a previous history of anginal seizures

Embolism must be considered in the differential diagnosis The severity of the pain and the absence of any condition that might serve as a prob able site of origin for an embolus are important points in drawing this distinction

## TREATMENT

Morphine and rest constitute the only therapeutic measures of value when the presence of this condition is suspected All unnecessary move ments are to be avoided in order to delay final rupture of the aneurysm through the adventitial layer If the rupture occurs back into the aortic lumen with the formation of a double aorta long survival may be possible in some instances The importance of rest in these rare cases until healing takes place is evident

## ILLUSTRATIVE CASES

### Hypertensive Cardiovascular Disease—Dissecting Aneurysm—Autopsy

**Case 50** N R a colored laborer of 44 was admitted to the Philadelphia General Hospital complaining of severe pain in the chest and abdomen

History The patient was well until a week before admission when he suddenly developed vertigo which was followed at once by a severe plitting pain in the abdomen and chest lasting several hours His right leg be ame numb following the seizure A few days later there was recurrence of pain and the patient was admitted to the hospital

Physical Examination Well nourished negro slight dyspnea B P 190/130 Advanced arteriosclerotic changes in both fundi Heart slightly enlarged to the left. The area of supracardiac dulness was increased There were systolic murmurs over the aortic and mitral areas Epigastric tenderness

Laboratory Data Wassermann negative W B C 16 000 The urine showed a light cloud of albumin but no casts

Course On the second hospital day the patient complained of severe chest and abdominal pain and died suddenly

Clinical Diagnosis A Etiologic Hypertension B Anatomic Di ecting aneurysm Slight cardiac enlargement C Physiologic Normal s nus rhythm D Functional Classi fication Class 4 Therapeutic Classificat on Class E

Autopsy Heart weight 450 Gm The heart (Fig I ) was moderately enlarged The valve rings were the seat of mild arteriosclerotic change The coronaries were slightly thickened The aorta contained a few calcified plaques Situated in the center of the posterior wall just distal to the aortic ring there as a match head ized rupture The media was split in its entirety involving the thoracic and abdominal segments to include the proximal 5 cm of the right common iliac The left renal artery was al o involved in the pro ess

## Hypertensive Cardiovascular Disease—Dissecting Aneurysm of Aorta—Survival for Five Months—Autopsy

**Case 51** A S an unemployed colored male of 60 was admitted to the Philadelphia General Hospital on 11/18/36 complaining of vertigo weakness and precordial pain



Fig 1 Dissecting aneurysm of aorta Note split in media (marked by arrow) (Autopsy No 30 3 6 Philadelphia General Hospital)

History High blood pressure for many years In 1931 he had a stroke but was able to be about again in a few months There was weakness of the left arm and leg

On ?/6/30 a sudden severe precordial pain appeared and lasted for two days It was relieved by rest in bed and medicine but returned although much less severe on several subsequent occasions Following one of the attacks the patient was admitted to the hospital

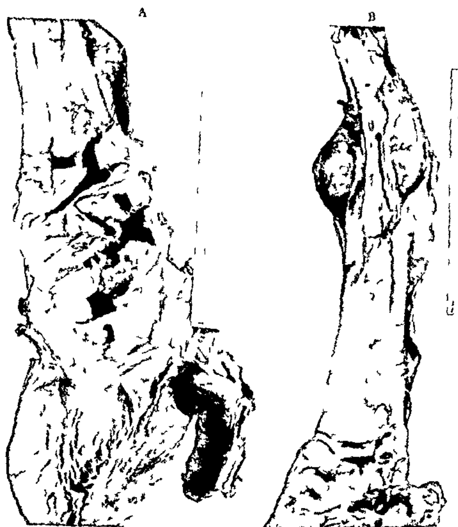



Fig 1 3 Dissecting aneurysm of the aorta A Point of rupture above aortic valve B Double aorta There were two separate channels as far as the bifurcation (Autopsy No 3 5?5 Philadelphia General Hospital)

Physical Examination Emaciated negro Slight dyspnea Old hemiplegia BP ?10/90 Pulse 84 totally irregular Heart markedly enlarged to the left on percussion Systolic murmurs were heard over the apex and aortic areas

Laboratory Data Roentgenogram hypertrophy of the left ventricle and widening of both ascending and descending portions of the aorta but no definite evidence of aneurysm

Electr ardio ram Auricul r fibrill ition
Wass rmann negative
CLINICAL DIAGNOSIS A Etiologic Hyp rt n B Anatomic Cardiac hyp rtrophy Aortic dilatation Coronary sclerosis Coronary occlus on C Physiologic Auricular fibrillat on Anginal syndrome D Functional Classification Class 3 Therapeuti Classi fication Class E

COURSE Many attacks of chest pain of the anginal type occurred from 1 /10/36 to 1?/15/36 The patient died suddenly during a severe seizure on 1 /15/36

AUTOPSY Heart weight 4 0 Gm Hypertrophy of the left ventricle with numerous fibrous scarred areas near the ap x There were atheromatous changes seen in the aorta and along the course of both coronary art ries (Fig 1 3) About 3 cm above th aort c cusp on the anterior aspect of the ascend ng branch th e as an old break in the intima leading into a separate healed channel ext nd ng to the bifurcat on (Fig 1 3) This new lumen was entirely covered by an endothelial surface The di sect on vas likev ise seen to extend upward into the innominate artery form ng two separ te lum na The d s-sected path vay opened into the right common carotid artery

# 10

# CONGENITAL HEART DISEASE

Congenital defects are rare and comprise less than 10 per cent of the cases in children's heart clinics and less than 2 per cent of all cardiac abnormalities seen in practice If these defects were listed with all their possible combinations this type of heart disease would immediately appear to be very complex Rather than wade through the intricacies of diagnosis presented by such an array it would seem easier to be satisfied with the plain diagnosis of congenital heart disease and allow the pathologists to worry about the difficulties of nomenclature and classification However during recent years the application of the newer laboratory methods and diagnostic procedures* to the problems of congenital heart disease permits a much sharper differentiation While an exact anatomic diagnosis is still a clinical impossibility in every case all patients should be carefully studied and the attempt made to record the defect that seems to be consistent with the physical signs

Aside from increasing our efficiency in cardiac diagnosis more exact knowledge of the structural defect is very useful in prognosis and in planning the patient's regime For example recently in a limited number of cases diagnosis of uncomplicated patent ductus arteriosus paved the way for the first successful ligation of this structure by Gross (page 339)

## ETIOLOGY

The chief cause of congenital heart disease is arrest of development and the earlier this occurs in embryonic life the more serious will be the resulting defect The critical time appears to be between the fifth and the eighth week just before the septa have come into apposition and the torsion of the great vessels is complete Interference in development during this period of growth will usually lead to a series of anatomic adjustments or rearrangements and multiple defects of a serious nature (morbus caeruleus) These are usually adaptations on the part of the growing embryo to permit survival in spite of grave alterations in the circulation

Interference with development at a slightly later stage before the septa *have closed (eighth week) produces localized septal defects* (cyanose tardive) while factors influencing growth after septal closure result in minor anomalies

Early writers pointed out the resemblance of many of these structural lesions to the hearts of animals and were content with the explanation of reversion to the primitive Some examples of a two-chambered heart which

* Particularly fluoroscopy

is similar to the heart of a fish appear and occasionally a three-chambered heart similar to that observed in the frog may be observed

Why this faulty development in the embryo occurs has not been satisfactorily explained but several theories have been advanced Some believe that abnormal currents in the fetal blood stream are present and prevent the septa from growing across and closing off the chambers in normal fashion Others believe that actual disease of fetal structures in the form of syphilis or tuberculosis is responsible The factor of heredity must also be given some credence since many cases live long enough to transmit the tendency Influences on the mother during pregnancy like fright operations etc are favored by the laity in the explanation of these anomalies as well as congenital defects of all types Inherent weakness of the germ plasm because of consanguinity and alcoholism must not be overlooked when we are searching for a cause in individual instances An early fetal endocarditis has been claimed to play a direct part in the formation of some of these defects but considering the evidence this seems to be a rare cause

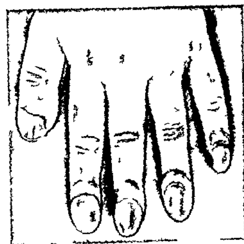
## SIGNS AND SYMPTOMS

At birth a loud murmur may be heard over the precordium If cyanosis is present a diagnosis of congenital heart disease appears quite certain However it is rarely possible at such an early date to classify the abnormality correctly If the child survives the picture becomes more clear cut and observations of the physical signs (thrills murmurs and cardiac hypertrophy) and study of the electrocardiogram and roentgenogram permit a more accurate diagnosis This does not mean that the signs present at birth should not be noted While a child s health record kept faithfully year by year is always a great asset in later life it is particularly valuable if evidence of a cardiac defect is present at birth Prolonged discussion concerning the etiology of the type of cardiac abnormality is then not so apt to arise when the murmur is heard for the first time after an attack of scarlet fever or other infectious disease of childhood As a rule murmurs heard in children under two years of age may be put down as congenital since acquired lesions prior to this age are rare

Cyanosis may or may not be present in congenital heart disease Large defects in the septum allowing considerable unaerated blood from the right side of the heart to mix with arterial blood on the left side produce cyanosis Since pressure is usually greater on the left side of the heart in small defects the direction of the flow will be from the left to the right Under special conditions however when the pressure in the pulmonary circuit becomes higher the flow may be from the right to the left for example in pneumonia after left ventricular heart failure or severe exertion This reversal of flow through the defect may cause a transient cyanosis In some congenital lesions where there is an interference with the entry of blood into the lungs (pulmonary stenosis) this additional factor contributes to

the cyanosis Cyanosis appears when the reduced hemoglobin exceeds the threshold value of 6 7 volumes per cent

Patients who have cyanosis soon develop clubbing of the fingers and toes (Fig 124) This is known as hypertrophic pulmonary osteoarthropathy (Marie 1891) While long recognized as an important sign in congenital heart disease it also occurs in subacute bacterial endocarditis and chronic suppurative conditions of the lung Infection plus obstruction to venous return seem in most cases to be the causative factors in initiating this hyperplastic process in the periosteum In congenital heart disease the clubbing of the fingers is present in direct proportion to the degree of cyanosis



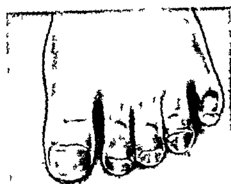


Fig 124 Clubbing of the fingers and toes

Polycythemia accompanies cyanosis and explains some of the symptoms commonly encountered in congenital heart disease When cerebral blood channels become clogged by the excess number of red blood corpuscles the patient may complain of headache and vertigo Syncope convulsions and even paralysis may follow when the degree of cyanosis and secondary polycythemia is extreme The latter symptoms are most apt to accompany a diminishing reserve and a secondary slowing of the cerebral circulation

Polycythemia results from an overactivity of the blood forming sites in their attempt to compensate for deficient oxygenation of the tissues The number of red blood cells in some cases may be increased to 12 000 000 per cubic mm The total blood volume and blood viscosity are also increased in these patients A stunted growth as well as abnormal cerebral development is not an unusual sequel in the presence of these marked circulatory defects

Symptoms of congestive cardiac failure may appear and complicate congenital heart disease at any time The incidence of pulmonary tuberculosis is also high All degrees of dyspnea may accompany congenital heart lesions, depending on the severity of the defect the amount of cyanosis

present and the activity of the patient In some defects paroxysms of dyspnea may follow exertion because of a temporary increase in the extent of the venous shunt

## CLINICAL CLASSIFICATION OF CONGENITAL CARDIAC DEFECTS

Since a variety of combinations of lesions is possible most classifications of congenital cardiac defects are lengthy and intricate There is little to be gained by attempting to remember all the types for even the more common ones are rare in the experience of any physician From the clinical standpoint it is sufficient to remember that these lesions may be conveniently grouped according to the presence or absence of cyanosis The congenital defect may offer a simple mechanical obstruction to the blood flow but no communication may exist between the pulmonary and systemic circulations Under these circumstances there is no cause for cyanosis In the next group defects in the cardiac or aortic septa may be present, but as long as the pressure remains higher in the systemic circuit blood passes from left to right and no cyanosis occurs If however the pressure is raised on the right side cyanosis appears (cyanose tardive) In the last group of cases a localized septal defect may be complicated by other anomalies which tend to raise the pressure on the venous side or one or more of the cardiovascular septa may be absent or the great trunks may be transposed A venous arterial shunt results in the development of permanent capillary changes and cyanosis Some of these cases may show a mechanical obstruction to the return of venous blood to the right heart which will cause a slowing of the capillary flow and cyanosis will result from stasis and increased de-oxygenation at the periphery

When attempting to demonstrate that cyanosis in children may not always have a cardiac origin I depend on the story of Dr J H Means of the negro mammy who refused to alter her technic and use a thermometer to test the temperature of the white child s bath Ah can tell by de chile said she *If de* water am too hot de chile he turn red if de water am too cold de chile turn blue

Based on the above considerations Abbott[1] proposes the following classification for cardiac defects The entities marked with an asterisk I have encountered in practice and consequently I am able to summarize their outstanding features in the following pages

Table VIII

ABBOTT'S CLASSIFICATION OF CARDIAC DEFECTS*

I ACYANOTIC GROUP

(No abnormal communications between the two circulations)
Pericardial Defects
Ectopia cordis
* Congenital idiopathic hypertrophy
Congenital aortic and mitral stenosis
Bicuspid aortic and pulmonary valves
* Supernumerary aortic and pulmonary cusps
Double A V orifices
Coarctation of the aorta of adult type
Pulmonary dilatation
Hypoplasia of aorta
* Double and right aortic arch
Left coronary from pulmonary artery
Congenital arteriovenous aneurysm
* Dextrocardia

II CASES OF ARTERIOVENOUS SHUNT WITH POSSIBLE TERMINAL OR TRANSIENT REVERSAL OF FLOW (CYANOSE TARDIVE)

Patent ductus arteriosus
* Defects in interauricular septum
Patent foramen ovale defects in upper and lower parts of the interauricular septum
Defects in aortic septum
Congenital aneurysm sinus of Valsalva
Communications between the aorta and pulmonary artery
Defects in the interventricular septum
(Maladie of Roger)

III CYANOTIC GROUP CASES OF VENO ARTERIAL SHUNT (MORBUS COERULEUS)

A Right sided valvular lesions Fetal passages closed Stasis and increased de oxygenation in capillaries
1 Pulmonary stenosis with closed septa
2 Congenital tricuspid stenosis with closed septa
B Cases of permanent venous-arterial shunt
1 *Moderate cyanosis*
Pulmonary stenosis with patent foramen ovale
Complete absence of interventricular septum (Cor biatriatum triloculare)
Common auriculo-ventricular ostium
2 *Marked cyanosis*
Pulmonary stenosis interventricular septal defect dextroposition of the aorta and right ventricular hypertrophy (tetralogy of Fallot)
Pulmonary atresia with defect of ventricular septum and dextroposition of aorta
Cor biloculare with transposition of great trunks
3 *Extreme cyanosis*
Transposition of great trunks with closed ventricular septum ductus arteriosus and patent foramen ovale
Pulmonary atresia with closed interventricular septum ductus arteriosus and foramen ovale patent
Tricuspid atresia
Cor biloculare
Mitral atresia
Aortic atresia

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### Congenital Idiopathic Hypertrophy

Hypertrophy of the heart at birth in the absence of any intra or extra cardiac etiological factor More frequent in males Survival is generally six months to a year rarely longer

**Symptoms** Usually pallor dyspnea and cyanosis The course is down hill! Death may occur suddenly

**Signs** Cardiac enlargement in all diameters (Fig 125) A systolic mur mur may be present

**Roentgenogram.** Characteristic (Fig 125)

**Electrocardiogram** Of little value unless there are associated defects



FIG 125 Roentgenogram of a one month old infant Note marked cardiac hypertrophy Autopsy showed von Gierke s glycogen storage disease

**Present day Views** The discovery of other causes for congenital hyper trophy is decreasing the number of cases labelled 'idiopathic In 1929 von Gierke s glycogen storage disease (cardiomegaly glycogenica) was described Deposition of large amounts of glycogen in the heart as well as in other organs (liver kidneys brain) occurs Anterior pituitary dysfunc tion and consequent defect in the glycogen splitting ferment has been ad vanced by some investigators as a cause for this rare disease which repre sents a persistence of the conditions that characterize fetal metabolism Glycogen can be readily demonstrated in the organs by the use of Best s carmine stain

**Treatment** At present there is no treatment known to control this per version of metabolism that results in structural changes in so many organs

BICUSPID AORTIC AND PULMONIC VALVES SUPERNUMERARY PULMONIC CUSPS




FIG 126 A Bicuspid aortic valve Secondary bacterial endocarditis ulceration and perforation This patient a white male of 46 developed aortic regurgitation while under observation in the hospital This was followed immediately by congestive cardiac failure Death occurred five days later The clinical diagnosis was syphilitic cardiovascular disease although the Wassermann was negative (Autopsy No 4773 Philadelphia General Hospital )




FIG 126 B Bicuspid aortic valve In this case the aortic valve was the seat of extensive deposits of calcium A terminal subacute bacterial endocarditis is seen (Autopsy No 33661 Philadelphia General Hospital )




FIG 1 6 C Supernumerary pulmonary cusps

1 Bicuspid Aortic Valve This defect per se does not produce any signs or symptoms However it is not without significance clinically since these valves very frequently become the seat of sclerotic change subacute bacterial endocarditis or both (Figs 126A and 126B) In some of these cases the aorta immediately above the valvular defect may show dilatation, dissection, and secondary rupture

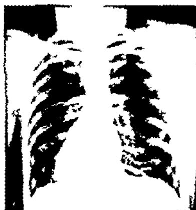
2 Supernumerary cusps are more frequently encountered at the pulmonary orifice where they are not often the seat of invasion and consequently are of little clinical significance A quadricuspid aortic valve is of more importance owing to the likelihood of the development of subacute bacterial endocarditis

## Congenital Dextrocardia

1 True dextrocardia (situs inversus) The heart is on the right side and the other viscera are likewise transposed

2 Dextrocardia without transposition of the other viscera is rare Its association with other anomalies generally determines the prognosis in each instance

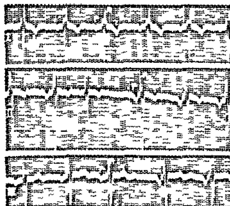
A



Clinical Diagnosis

Possible if palpation and percussion routinely practiced to the right as well as to the left of the sternum

B



All waves in lead 1 are reversed in direction Note that lead 3 is equivalent to lead 2 and 2 to 3

Fig 127 A Congenital dextrocardia B Electrocardiogram of patient who had congenital dextrocardia and acquired mitral stenosis (see Fig 77)

Dextrocardia is unimportant if no other cardiac defects co-exist It does not shorten life There are no symptoms

### Interventricular Septal Defect (Roger's Disease)

**Usual Site** High up in the fibrous part of the septum (See Fig 132)

**Direction of Blood Flow** From left to right Reversal of flow may take place during terminal stages with cyanosis

**Heart** Enlargement may be present or absent Murmur is loud, harsh *and systolic in time Point of maximum intensity is the third or fourth* intercostal space close to the sternum A thrill is palpable over this area

**Electrocardiogram** In rare cases the defect involves the conduction system and causes heart block (page 345)

**Prognosis** Depends on associated defects and heart size The danger is subacute bacterial endocarditis (Case 87) Interventricular septal defect is one component of Fallot's tetralogy (page 347)

Note If the coronary artery supplying the interventricular septum becomes occluded in later life and the infarct softens and ruptures an acquired interventricular septal defect may appear It is usually small and unimportant but may produce physical signs similar to the congenital type

### Coarctation of Aorta

**Types** 1 Infantile Narrowing of the isthmus of the aorta between the left subclavian and the ductus arteriosus (Rare)

2 Adult Narrowing of the aorta at the site of the ductus arteriosus (Common type)

**Symptoms** Variable often none There may be intermittent claudication in the absence of demonstrable arterial disease

**Cardiac Signs** Hypertrophy Followed at times by signs of failure

**Electrocardiogram** Not characteristic

**Diagnosis** Not difficult if the possibility is kept in mind and the characteristic features are sought

#### Vascular Signs

Hypertension in upper extremities

Decrease in blood pressure in lower extremities Collateral circulation occurs through anastomoses of internal mammary and epigastric arteries and through dilated intercostal vessels (Fig 128C)

Tortuous or pulsating arteries may be seen or felt under the skin in posterior wall of the thorax around the scapula or in the anterior ab dominal wall

Thrills and murmurs may appear along the course of these vessels

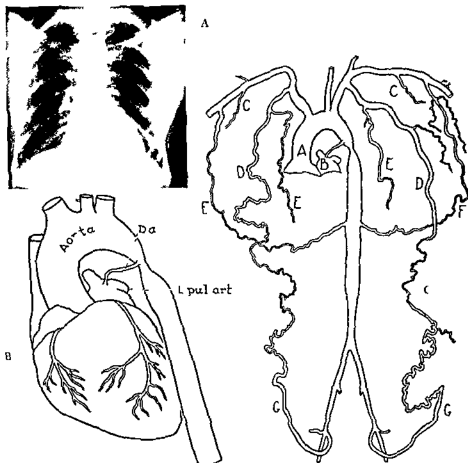



Fig 1 8 A Roentgenogram in coarctation of the aorta Characteristic scalloping of the inferior margins of the ribs se n This is caused by eros on of bone by dilated inter o tal a teries (Courtesy of Dr Paul A B shop ) B Drawing of coarctation of th ao ta of the adult type Note marked narrowing of the aorta just below point of in s rtion of d ctus arte iosus C Collateral circulation in a case of coarctation of the aorta (A Aorta B Pulmonary artery C Subscapular artery D Internal mammary artery E Thyroid artery F Int rco tal artery G Ep gastr c artery )

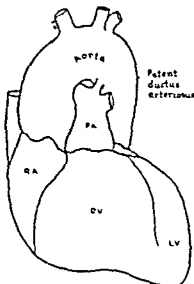
## Patent Ductus Arteriosus (Commonest congenital defect)

**Etiology** The ductus may remain open to compensate for the other defect (coarctation) consequently it is less often found uncomplicated

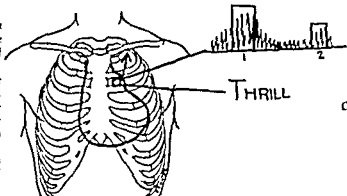
**Direction of Flow** Pressure in the aorta is higher therefore the flow is from the aorta to the pulmonary artery (arteriovenous shunt)

**Symptoms** Usually none

A 



B



C

Fig 1 9 A Roentgenogram in patent ductus arteriosus Note the great dilatation of the conus pulmonal This is more extreme than that noted in mitral stenosis Some enlargement of the heart to the left is also seen B Diagram showing patent ductus arteriosus C Chart of physical findings

### Signs

Continuous murmur and thrill at the second left interspace near the sternum

The heart shadow in marked cases shows enlargement of the pulmonary artery, and hypertrophy of all chambers of the heart may be present

Increased pulse pressure and the peripheral signs may be present as in aortic regurgitation In the case of patent ductus arteriosus the regurgitant stream goes into the pulmonary artery

**Prognosis** Depends upon the size of the communication Surgical ligation is possible in selected (uncomplicated) cases (page 339)

In fetal life the blood is short-circuited around the lungs from the pulmonary artery to the aorta through the ductus arteriosus (Fig 129B) When the lungs expand after birth the ductus normally closes and becomes atrophic If it remains open the blood flow through this structure is reversed

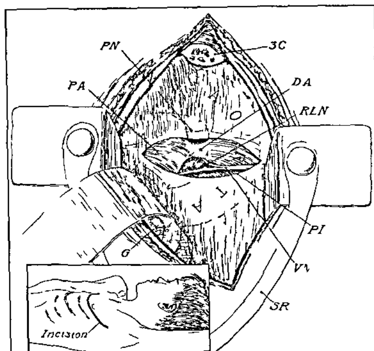



FIG 130 Sketch of operative exposure of the ductus hich was 11 mm in d ameter Insert shows position of the patient w th the left arm drawn up along the head and with incis on coursing just beneath the breast Thorax entered through the third inter space Third cartilage cut to allow upward retraction of ribs The left lung s held down infer orly w th a gauze pack and malleable retractor Positions of aorta and pulmonary artery indicated by dotted l nes When the pleural covering of the medias t num was incised a direct view of the aortic arch pulmonary artery and ductus was obtained (3C) Cut end of third co tal cartilage DA Ductus arteriosus G Gauze pack over collapsed lung PA Pulmonary artery PI Pleural incis on PN Phren c nerve RLN Recurrent laryngeal nerve SR Self retaining retractor VN Vagus nerve

since the pressure in the aorta is greater than in the pulmonary artery If the passageway is large the effect on the circulation is the same as that of any other arteriovenous aneurysm or shunt About two-thirds of the patients who have a patent ductus arteriosus show an associated anomaly but in one third of the cases the patent ductus exists as the only defect Recently the latter group has assumed considerable importance since Gross has demonstrated that this arteriovenous communication can be succe fully ligated (Fig 130)

While in some instances uncomplicated patent ductus arteriosus may be compatible with a normal life, in Abbott's series of 92 patients showing this defect, 2 died of subacute bacterial endocarditis, 24 of cardiac decompensation, 16 of sudden heart failure, and two of sudden rupture of the ductus. The average age at death was 24 years. Consequently the reason for employing surgical measures in these selected instances is to improve a most uncertain future. The patient who has an uncomplicated patent ductus arteriosus faces the danger of developing subacute bacterial endocarditis or endarteritis at any time. If this complication is avoided, the possibility remains that the shunt may increase in size and cause cardiac hypertrophy and subsequent failure. In a few cases the patent ductus may dilate and become thin walled, in which event the danger of rupture is not negligible. These ever present threats are eliminated by ligation.

Before recommending operation, the presence of associated defects such as aortic stenosis or coarctation, stenosis or atresia of the pulmonary artery, or the complication of bacterial endocarditis should be ruled out. Pulmonary stenosis may offer some difficulty in the differential diagnosis, although the presence of a loud pulmonic second sound in cases of patent ductus and its absence in disease of the valve is an important point in drawing the distinction. In pulmonary stenosis, a right axis deviation appears in the electrocardiogram, while cardiac hypertrophy and cyanosis are more apt to be present. Associated anomalies like septal defect or a mitral stenosis, if slight, are not contraindications to operation, for as Cross points out, some improvement may follow if one defect is corrected. Vegetations of a subacute bacterial endocarditis present near the opening of the ductus arteriosus may be dislodged at the time of the operation; consequently their presence should be regarded as a complication.

In some cases where patent ductus arteriosus is the single lesion, spontaneous closure in later life may take place. If the findings are at all suggestive of this occurrence, operation should not be considered. In the absence of cardiac enlargement in patients who have a normal diastolic pressure and only a slight fulness in the region of the pulmonary artery on fluoroscopy, operation likewise has little to offer. When the heart is increasing in size and the physical findings are becoming more pronounced as the child grows older, operation is indicated.

## SUMMARY OF CRITERIA FOR OPERATION (CROSS)

1. Presence of machinery murmur in pulmonary area, accompanied by increase of P2, most intense in the same area.
2. Congestion of the lung fields in the roentgenogram.
3. Prominent pulmonary artery in roentgenogram.
4. Roentgenologic evidence of cardiac enlargement, particularly in the region of the left ventricle.
5. Systolic blood pressure normal with a lowered diastolic level.

The operation recommended and successfully carried out by Cross

exposes the ductus from the left side The pleural cavity is entered, the lung collapsed, and the structures in thi manner adequately exposed (Fig 130) Gross reports little postoperative reaction no important postoperative complications and no mortality in his first four cases In each patient following operation the thrill disappeared and the diastolic pres ure ro e to a normal level In two instances the transverse cardiac diameter decreased and one undernourished child gained nine pounds following ligation After the procedure each child returned to school with a much improved circulation

INTERAURICULAR SEPTAL DEFECTS

**Entire absence of auricular septum** may occur but is very rare (cor triloculare biventriculare) (page 343)

**Persistent ostium primum** follows arrested development of septum primum *and is seen in the lowest part of the interauricular septum* (Fig 131)

**Patent foramen ovale** is more frequently encountered A valve like opening is seen in 25 per cent of autopsies but is unimportant When the foramen ovale remains widely open because of a defect in the ostium secundum it is significant This defect often accompanies persistent ostium primum

If not associated with other defects there may be *no signs or* symptoms Subacute bacterial endocarditis *is rare in this region* but paradoxical embolism may occur Thrombi from veins pas ing through the interauricular septal defect tend to lodge in the systemic circulation Emboli may also go from the left auricle to the lung through this septal opening

Case 52 (Fig 131) A poorly nourished white female baby six months of age was admitted to the Philadelphia General Hospital for bronchopneumonia Examination of the heart was entirely negative Death occurred on the eleventh hospital day following the complication of acute otitis media Cyanosis was present shortly before death

AUTOPSY showed interauricular septal defect as the only congenital anomaly Death was caused by bronchopneumonia acute otitis and sepsis



FIG 131 Int au icular septal defect (Autopsy No 4 099 Philadelph a Gener l Ho pital)

### Patent Foramen Ovale Defects in Interventricular Septum Autopsy

**Case 53** An emaciated colored female child of four was admitted to the Philadelphia General Hospital complaining of shortness of breath and cough of four months duration

Physical Examination showed precordial bulging precordial thrill over the third left interspace and a loud rough systolic murmur in the same region The liver was two fingers breadth below the costal margin Wassermann negative

Autopsy Cardiac hypertrophy Patent foramen ovale Three separate openings between the left and right ventricles were discovered in the upper portion of the septum (Fig 152) No other defects



Fig 152 Interventricular septal defects (Autopsy No 364 Philadelphia General Hospital)

## ILLUSTRATIVE CASES

### Complete Absence of Interauricular Septum and of the Membranous Portion of the Interventricular Septum—Congenital Heart Block—Autopsy[04 06]

**Case 54** An infant boy age six hours th sixth child of a woman age 8 was admitted to the Woman s College Hospital on May 6 193 The father and mother and the other ch ldren were free of congen tal defects The mother s prenatal period was entirely uneventful and the del very was norm l The Wassermann reaction of the mother s blood was negative

On admission the infant was well developed and well nourished and as apparently a f ll term baby No defects were noted The weight was eight pounds (3 630 Gm) There was inten e cyanosis of the body generally and of the lip ears and nails especially Examination of the lungs show d mpaired resonance at the left apex poster orly The breath sound were harsh over both lung fields and there were many crep tant rales throughout Substernal inspiratory retraction was present The left border of the heart was 7 cm from the mid ternal line in the fourth interspace The right border was 5 cm from the m d te nal line in the fourth nterspace The fir t sound was obscured by a blo ving systolic murmur and the second sound was eak The murmur was audible over the nt re chest but th center of intensity was in the second interspace at the left of the sternum The rate of the heart beat was 40 per minute There ere no thrills The liver and spleen were not palpable and the rest of the examinat on was negative

The child was taken to the heart stat on of the hosp tal where electrocardiograms w re made at intervals for the next fe hours All tracings showed complete heart block with the ventr cular rate of 50 the auricular rate of 100 and low voltage (Fig 133B) Oxygen was administered ith sl ght improvement in the cyano s The electrocardiogram was not alte ed

The ch ld wa taken back to the ward and kept in an oxygen tent A roentgenogram of the chest (Fig 133A) showed the heart shado to be very large in all diamete The child d ed 18 hours after it b rth

Diagnosis A Etolog c Cong nital defect B Anatomic Card ac hypertrophy Interv ntricular septal d fect C Phys ologic Complete heart blo k D Funct onal Class fication Class 4

Autopsy The main defect of the heart was th al no t complete ab nce of the nter a r cular septum and the membrano port on of the terventri ul r septum The coronary veins em pt ed separat ly into the m ddle of th po ter o wall of the common auricle The venae cavae had a common openi g The m t al and tr cu p d valves were completely fo med and were attache l m d ally to the upper edge of the interventricular septum The aorta and pulmonary arte y had approximately normal relat onsh p

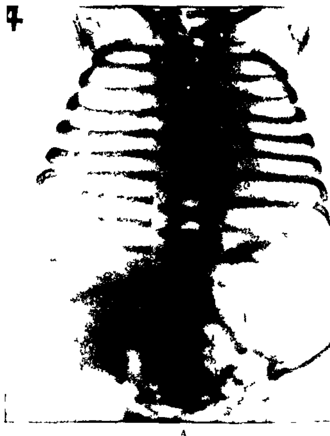
Microscopically t was obse ved by means of serial sect ons that there was practically complete abse ce of the conduct on bundle between the common auricle and the ventricles The d fect had completely severed the aur culove itr cular nod from the auriculo ventricular bundle Th bundle of H s and the left bundle branch were well developed but a definite r ght bundle branch ould not be found although there was a suggestion of one

Discussion In this case we have an example of a three chambered heart with one auricle and two ventricles (cor triloculare biventriculosum) Other cases of heart block of congenital origin associated with this anomaly have not been reported

A complete block of the contraction impulse from the auricle occurred in the upper third of the interventricular septum at the site of the defect

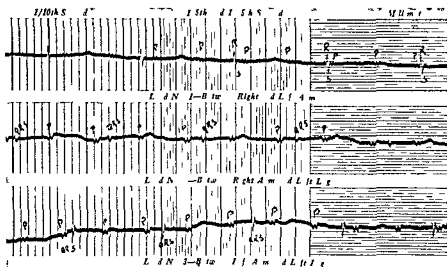
This anatomic separation was shown to be of developmental and not of inflammatory origin by serial sections The bundle of His was well formed but was disconnected from the auriculoventricular node in the region of the defect

The clinical diagnosis of congenital heart block is not difficult When the presence of auriculoventricular dissociation is proved by an electrocardiogram taken shortly after birth a defect in the upper third of the interventricular septum is at once suggested In this patient the cyanosis cardiac

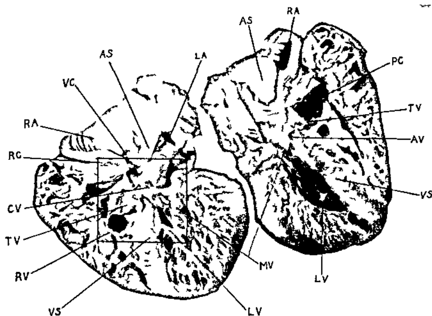


A

Fig 133 A Roentgenogram taken four hours before death Note enlargement of the heart in all diameters B Electrocardiogram Complete heart block is present The auricular rate is 100 and the ventricular rate is 50 C The interior of the heart bisected in sagittal plane A the posterior half; B the anterior half VS interventricular septum RV right ventricle; TV tricuspid valve; CV openings of coronary veins in auricle; RC right coronary artery RA portion of the common auricle corresponding to normal right auricle; VC common orifice of vena cavae; AS spur in roof of common auricle dividing it incompletely into right and left halves; LA portion of the common auricle corresponding to normal left auricle; *MV* mitral valve; *LV* left ventricle PC pulmonary conus, AV aortic orifice under anterior leaflet of the mitral valve



B




C

enlargement and the systolic murmur were physical signs in keeping with this impression The clinical recognition of the other abnormalities associated with the interventricular septal defect in this case was, of course, impossible

The diagnosis of congenital heart block in older children in addition to the typical electrocardiogram rests upon the history of bradycardia at a very early age and the absence of an infection that might cause heart block after birth such as rheumatic fever chorea diphtheria or congenital syphilis The occurrence of syncopal attacks at an early age is fairly good evidence of the existence of heart block prior to the attacks

### Pulmonary Stenosis

Complete atresia may be present (page 348) or the pulmonary valve may be markedly stenosed the pulmonary artery small and the right ventricle large Associated septal defects are very common because of obstruction of the blood flow from the right ventricle and are in some measure compensatory Pulmonary stenosis is a component of Fallot s tetralogy (page 347) In complete atresia the blood may reach the lungs through a patent ductus arteriosus If this is absent dilatation of the bronchial arteries usually occurs Uncomplicated pulmonary stenosis is rare

**Signs** Cyanosis stunted growth clubbed fingers polycythemia thrill and harsh systolic murmur in the second or second and third intercostal spaces just to the left of the sternum The pulmonic second sound is diminished or absent

**Electrocardiogram** Marked right axis deviation is encountered in addition to the large P waves that are associated with right auricular enlargement (Fig 135)

### Tetralogy of Fallot

(Most common cause of congenital cyanosis in patients surviving to adult life)

The combination of defects that makes up the tetralogy consists of

1 Ventricular septal defect
2 Pulmonary stenosis
3 Dextroposition of the aorta
4 Hypertrophy of the right ventricle

If pulmonary stenosis develops before the septum closes blood is shunted from the right to the left side of the heart and then to the aorta which is dextroposed This is followed by hypertrophy of the right ventricle Cyanosis is always present

Signs A systolic murmur is present over the precordium and is most intense over the pulmonary area There is a systolic thrill in the pulmonary area

Electrocardiogram Right axis deviation

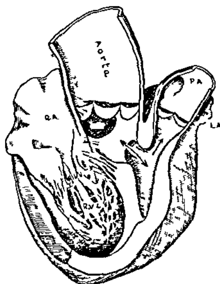



Fig 134 Schematic drawing illustrating Tetralogy of Fallot

Roentgenogram Diminished prominence of the pulmonary conus with elevation of the apex by enlargement of the right ventricle may give coeur en sabot appearance (Figs 29 and 136)

### Pulmonary Stenosis Associated with Patent Foramen Ovale—Autopsy

**Case 55** A white American male 22 years of age was admitted to the Tuberculosis Division of the Philadelphia General Hospital complaining of cough hemoptysis and dyspnea Extensive tuberculous invasion of both lung apices was found and the sputum was positive for tubercle bacilli There was a systolic thrill and murmur over the pulmonary area The fingers were clubbed Cyanosis was present

Autopsy Heart weight 420 Gm The pulmonic valve had two cusps and showed marked stenosis Foramen ovale patulous No other defects

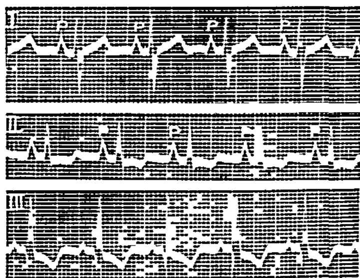



Fig 135 The electrocardiogram in congenital pulmonary stenosis Note high pointed P waves in the first two leads Right axis deviation is present

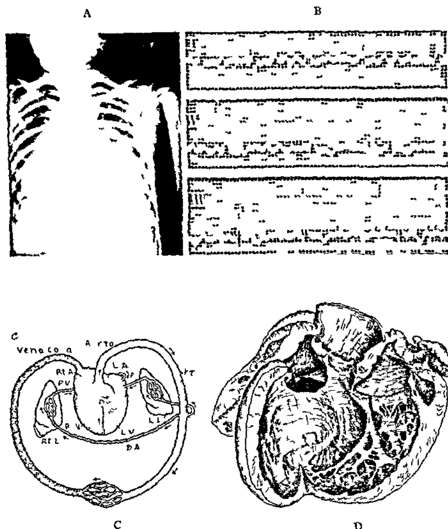



*Fig* 136 A Roentgenogram of chest Note "coeur en sabot" appearance of cardiac silhouette There is considerable enlargement of all chambers B The electrocardiogram Auricular fibrillation is present Right axis deviation C Diagram illustrating circulation in Case 56 The aorta is dextroposed The bronchial arteries (B A) supply the lungs They arise from the descending aorta at the level of the trachea (T) There is complete pulmonary atresia Interventricular septal defect is also present D Drawing of specimen Note hypertrophy of the right ventricle complete pulmonary atresia dextroposition of the aorta and interventricular septal defect

## ILLUSTRATIVE CASE

### Complete Pulmonary Atresia with Defect of Ventricular Septum Dextroposition of the Aorta and Hypertrophy of the Bronchial Arteries—Autopsy [19]

**Case 56** A cyanotic male child of five was admitted to Memorial Hospital complaining of shortness of breath and vomiting. He was well until *four months* before admission when he had a sudden convulsive seizure following exercise. Dyspnea fatigue and cyanosis appeared for the first time. A second convulsive seizure the day before admission was followed by edema dyspnea and vomiting.

Physical Examination BP 90 60 Pulse 1 0 totally irregular Cyanosis Dyspnea Edema of the extremities to the knees Marked bulging of the left side of the chest Apex beat in the anterior axillary line in the sixth interspace There was a thrill palpable over the third intercostal space to the left of the sternum A systolic murmur was heard over the whole precordium loudest in the region of the thrill Systolic and diastolic murmurs were present over the cardiac apex The liver edge extended to the umbilicus No ascites. *Dulness of the right chest posteriorly to the scapular angle Fingers and toes* showed marked clubbing (See Fig 1 4)

Laboratory Data Blood count hemoglobin 1 0 per cent (Sahli); R B C 6 410 000; W B C 6 700; Differential normal Wassermann and Mantoux tests negative The urine showed a light cloud of albumin and 3 R B C per H P F

Electrocardiogram (Fig 136B) auricular fibrillation and right axis deviation

Roentgenogram (Fig 136A) heart enlarged in all diameters Note coeur en sabot" outline A concavity replaces the curve in the left border usually formed by the pulmonary artery

Clinical Diagnosis A Etiologic Congenital Tetralogy of Fallot (?) B Anatomic Cardiac enlargement Interventricular septal defect pulmonary stenosis right sided aorta C Physiologic Congestive cardiac failure Auricular fibrillation. D Functional Classification Class 4 Therapeutic Classification Class F

**Discussion** Bed rest and digitalization resulted in slight improvement Three days after admission there was a sudden attack of dyspnea progressing *to orthopnea during which the cyanosis became extreme* and marked distention of the neck veins appeared A venesection (300 cc ) was performed and the patient was placed in an oxygen tent Theoretically oxygen administration should not be expected to relieve the cyanosis of congenital heart disease However some improvement was noted in this instance The digitalis was cut to a maintenance level of 2 grains daily on the eighth hospital day The liver appeared to be much smaller in size During the third week in the hospital death occurred suddenly following a third convulsive seizure

This patient shows that little decrease in the exercise tolerance is possible in some instances in the presence of marked derangement in anatomic structure This lad lived on the third floor of a city apartment house where there was no elevator It is remarkable that he was able to climb these steps many times daily without the appearance of cardiac symptoms Unfortunately, he was also allowed to engage in active play with other children and this contributed to the rapid breakdown of his reserve The slowing of the cerebral circulation in the early stage of failure in the presence of the polycythemia precipitated the convulsive seizure Following this the decline

was rapid The persistent vomiting that preceded his admission to the hospital probably was caused by stasis of the circulation and the clogging of the blood channels along the gastro-intestinal tract

The congestive failure was treated in the same manner as failure caused by any other type of heart disease Death occurred following a convulsion and was attributed to cerebral thrombosis

Four other examples of complete pulmonary atresia with closed ductus arteriosus and a pulmonary circulation carried on through dilated bronchial arteries have been previously described in the literature The ages of death of these patients were 33 30 20 and 6 years all surviving longer than this patient Two of the patients in the series died following intercurrent infections, while thrombosis in the bronchial arteries was the cause of death in the other two The patient showing the smallest bronchial arteries died in childhood so it may be said that the larger the bronchial arteries in the presence of complete pulmonary atresia the better the chance of survival

The occurrence of auricular fibrillation in this patient is worthy of note for the arrhythmia is very rare under the age of 15 It is likewise seldom seen associated with congenital heart disease However while advanced cardiac disease of any type may be present without the occurrence of auricular fibrillation when congestive failure occurs with its auricular strain fibrillation may appear at any age

There is some excuse for mistaking complete pulmonary atresia for the tetralogy In the first place if the ductus arteriosus is closed the coeur en sabot appearance of the roentgenogram suggests the latter diagnosis when other manifestations of congenital heart disease are present When the interventricular septal defect is small and the murmur loud and well transmitted to the base of the heart differential diagnosis becomes most difficult if not impossible However in the absence of a murmur and thrill over the pulmonary area in patients showing this typical roentgen ray picture in addition to dextroposition of the aorta polycythemia and clubbing the diagnosis of complete pulmonary atresia is to be thought of If the patient is an adult greatly enlarged bronchial arteries can be predicted

## TREATMENT

When patients who have congenital cardiac disease survive the period of childhood the greatest threat to life is the development of subacute bacterial endocarditis at the site of the malformation In many of the cases the defects are otherwise compatible with a long existence and in this respect resemble healed rheumatic lesions Subacute bacterial endocarditis is most apt to be superimposed on interventricular septal or aortic defects patent ductus arteriosus or bicuspid aortic valves The blood flow through the abnormal shunts in time produces fibrosis and thickening about their margins and on the walls of the right ventricle and pulmonary artery

*These are ideal locations for the growth of micro-organisms Auricular* septal defects escape this danger Following tonsillectomy dental extractions or any other operative procedure when a temporary blood stream invasion is apt to occur sulfanilamide or one of its derivatives should be administered (page 196)

The treatment of congenital heart disease lies chiefly in the prevention of complications These children should be guarded against infections of all types in much the same manner as the rheumatic group Upper respiratory infections or focal infections in teeth tonsils or sinuses and all minor ailments should be carefully managed and always regarded more seriously in children who have congenital heart disease than in normal children Pulmonary tuberculosis is a danger particularly in patients who have defects of the pulmonary artery

Children who have serious defects and permanent cyanosis should never be allowed to attend public schools where the temptation is present to compete with normal children Special schools that provide individual attention are needed The acyanotic group may attend school but should not be allowed to engage in competitive athletic activities Exercises that do not produce cyanosis or dyspnea however may be allowed

Careful medical supervision is needed in order that the problems of *management may be intelligently met as they arise Children who have* congenital lesions should be frequently examined at the time of puberty, when rapid growth adds another burden to the circulation

Proper guidance during early years especially in the selection of a suitable occupation that will not place too great a strain on the circulation in later life or expose the patient needlessly to changing weather conditions is very important

In the cyanotic group the treatment can only be palliative since the oxygen unsaturation eventually creates a train of complications all of which tend to influence the course unfavorably Sudden death is not infrequent in many cases of this group It may also follow spontaneous rupture of the aorta or cerebral hemorrhage in patients who have coarctation When we consider the group of patients suffering from congenital heart disease as a whole the most common cause of death is found to be congestive failure (Case 56) Its management is the same as outlined for other types of heart disease with the exception of the fact that when congestive manifestations appear in congenital heart disease the outlook is exceedingly poor

## PROGNOSIS

The prognosis in congenital defects in the absence of cyanosis and clubbing is good as far as the lesion itself is concerned The complications however, are always a threat and therefore prognosis should be guarded If the only abnormality is patent ductus arteriosus operation offers a possibility of cure unless this lesion compensates for some other defect

# 11

# THE HEART IN THYROID DISEASE

The cardiac mechanism may be influenced by disorders having their origin elsewhere in the body Chief among these are disturbances of the thyroid gland which may be accompanied by either excessive secretion (hyperthyroidism) or a deficient secretion (hypothyroidism) In either of these conditions the cardiac symptoms may so overshadow the rest of the clinical picture that a diagnosis of some form of primary heart disease may be made Consequently successful management will depend on recognition of the correct etiologic background

## HYPERTHYROIDISM

Hyperthyroidism or thyrotoxicosis may appear in patients with the nodular type of goiter (toxic adenoma) or it may develop as a part of the classical picture of exophthalmic goiter In either instance the same secondary disturbances take place in the circulation In nonendemic areas the incidence of the thyrotoxic state depends upon the ability of the physician to recognize the early symptoms of the disease White[393] found hyperthyroidism to be the main factor in producing cardiac symptoms in 5 per cent of his patients

**The mechanism** by which thyroid secretion in excessive or toxic amounts affects the myocardium has been long debated Some authorities claim that it is merely an exhaustion state the end result of overaction of the organ induced by the elevated metabolic rate Others claim that a specific myocardial lesion is produced but much doubt remains concerning the existence of such a structural change Most of the abnormalities that have been described are merely incidental and do not appear to be specifically related to the thyroid hyperfunction

### Signs

There is a wide variation in the clinical picture produced in different patients by the same degree of thyroid overactivity The main symptoms palpitation and tachycardia may be present continuously when secondary to a sinus tachycardia or paroxysmal when arising from episodes of auricular fibrillation or flutter Palpitation may be mild at first appearing only after exertion Later the rapid heart rate that produces palpitation may be present at all times

In older patients who have already some degree of coronary sclerosis the added burden of thyrotoxicosis may be just enough to accentuate this underlying defect, and angina may appear

Some patients will show a quick progression of the disease with the early appearance of edema and other congestive manifestations In this group the thyroid background may be entirely overlooked in the presence of other coincidental factors such as hypertension or coronary disease It is therefore *most important to be continually on the lookout for this type of case,* for if the correct etiology is discovered proper treatment may restore the patient to normal activity for an indefinite period Some observers contend that these advanced symptoms of congestive failure are not produced by the action of the excess of toxic thyroid secretion on a normal heart but appear only in the presence of a previous cardiac lesion of rheumatic sclerotic or hypertensive type The hyperthyroidism in these cases acts merely as the precipitating factor These primary conditions, of course do not disappear following successful treatment of the thyroid disorder

## Pathology

When the pathologist views the thyroid heart at autopsy he can point to no specific lesions that characterize the condition Slight enlargement of the heart is the rule and there is usually an increase in the size of the individual muscle fibers but the various chemical changes responsible for the abnormal cardiac action elude detection No necrotic areas or inflammatory changes are encountered in the endocardium myocardium or pericardium

If we view the heart in this condition as only one organ of the body *that is influenced by a generalized disturbance the symptoms* will be better understood The increased metabolism speeds all bodily processes which naturally places a demand on the heart for more blood the amount being governed by the degree of elevation of the metabolism A normal heart can usually meet these demands indefinitely while a diseased organ will show hypertrophy and ultimately failure will appear Some observers[36] view the heart in hyperthyroidism as a circulatory problem quite similar to the arteriovenous aneurysm since all vascular channels in the thyroid are widely dilated

## Incidence

Hyperthyroidism may appear at any age The average reported by Hurxthal[105] is 37 while in a series reported by White and Jones[393] 56 per cent were found to be between the ages of 30 and 50 In all the groups studied women predominate although usually men show the more severe cardiac reactions While race has little influence on the incidence of thyrotoxicosis, the incidence reported among negroes is low

## Diagnosis

Examination of the thyrocardiac patient reveals an increase in the rate and force of the heart action The apex beat is usually readily visible and the palpating hand will detect an increased force of the cardiac impulse

An axillary impulse may be seen at times in the presence of an overactive heart and care should be taken that this is not interpreted as evidence of hypertrophy Increased pulsation of the vessels of the neck may also appear The entire picture of overaction is reflected in the cardiac silhouette at the time of the fluoroscopic examination and this observation serves as valuable suggestive evidence Systolic murmurs are not uncommon in thyrotoxicosis particularly over the region of the pulmonary artery while increase in the size of this vessel and in the entire pulmonary circulation may appear in advanced stages of the disease (see Fig 139)

Examination of the neck in most cases reveals some enlargement of the thyroid gland and the well known eye signs (exophthalmos inability to converge widening of palpebral fissures and lid lag) may be in evidence All of these may be absent and the patient may show only a peculiar staring expression which is just as valuable an observation The skin of the thyrocardiac patient is warm moist and flushed and quite characteristically elastic even though he may have lost 30 to 40 pounds in body weight The patient s attitude is alert and the motions are quick nervous and at times purposeless The pulse is rapid averaging about 110 The rhythm of the pulse at first is regular (sinus tachycardia) later becoming irregular owing to the presence of premature beats or paroxysms of auricular fibrillation or flutter These paroxysms may recur with increasing frequency until the arrhythmia usually auricular fibrillation becomes established The patients who show congestive failure quite often have auricular fibrillation as a complication

**Blood Pressure Change** The most constant change in the blood pressure is an increased pulse pressure caused by a fall in the diastolic level and a less constant elevation of the systolic reading Evidence of this increased pulse pressure is seen in the dancing of the arteries on physical examination and in the increased range of cardiac contractions on fluoroscopy The increased pulse pressure may give a Corrigan pulse and other peripheral signs

**The electrocardiogram** in thyrotoxicosis reveals the nature of any arrhythmia that may be present but it does not have a pattern that is characteristic of the condition High T waves are often observed but are not invariably present in fact T may at times be inverted An uncomplicated sinus tachycardia is the usual finding on electrocardiographic examination in hyperthyroidism

Determination of the **basal metabolic rate** is a reliable diagnostic procedure if carefully carried out by an intelligent technician More than one estimation should be made Usually the basal metabolic rate in thyrotoxicosis will be from 30 to 75 per cent above the normal Other laboratory tests may be of value in the diagnosis of thyrotoxicosis when doubt exists The blood cholesterol values tend to be definitely diminished in hyperthyroidism there is also a decrease in creatine tolerance [66] an increase in the rate of the circulation and the blood volume

In younger patients the diagnosis of thyrotoxicosis is not difficult when

the expression is staring and there is exophthalmos, tremor, sweating, and enlargement of the thyroid gland In elderly patients the condition is more apt to be overlooked since the gland enlargement may be absent or hidden beneath the sternum or the sternomastoid muscle the eye signs few and the flushing less noticeable A careful search however, may show a fine tremor

**The loss of weight** is usually marked even though the patient has a splendid appetite and consumes hearty meals The recognition of this fact serves to differentiate the condition from other wasting diseases The elderly patient with thyrotoxicosis who comes to the physician complaining of dyspnea chest pain weakness palpitation or edema is quite apt to have an associated arteriosclerosis It is important therefore to keep the possibility of thyrotoxicosis constantly in mind and to make a careful search for additional clues in older patients

**The signs of mitral stenosis** may be imitated by the overactive heart of thyrotoxicosis The first sound at the apex will be accentuated in both conditions but the thrill and the murmur in mitral disease are diastolic This usually serves to make the distinction However mitral stenosis may be present occasionally with superimposed thyroid disease (see Case 16) in which event other signs must be relied upon Digitalis will not so readily slow the rapid fibrillation that accompanies thyrotoxicosis and this observation often leads to the detection of the thyroid background The reduction in the pulse rate in thyrotoxicosis that follows the administration of iodine is another therapeutic test of value

### Course

The course of the thyrotoxic process in any patient depends on a number of factors age sex social position duration of the symptoms and the presence or absence of another form of heart disease The last circumstance may predispose to the early appearance of heart failure while hyperthyroidism in younger patients with normal hearts may be well tolerated for many years

### Treatment

Many forms of treatment have been recommended for thyrotoxicosis When cardiac manifestations are prominent and progressive however there is only one surgery Subtotal thyroidectomy brings quicker and more certain restoration of health and for this reason is the best procedure particularly in patients who have to earn their own living

Much has been claimed for the cures achieved by continuous medical management but these are uncertain and often hard to evaluate properly, since the course of the disease in many patients shows a natural tendency to remissions Many patients cannot afford to take the long rest periods that this course of treatment entails Surgical relief is eventually sought in most cases so in the long run when cardiac symptoms are present nothing is gained by delay Where the diagnosis is in doubt or where the

condition shows a tendency to run a very mild course sedatives rest and psychotherapy may prove beneficial

**Pre operative Treatment** Medical management however is most essential in the preparation of the patient for surgery Rest in bed and the administration of iodine in some form over a period of two weeks prior to operation constitute a regime of recognized value The form of the iodide is not a matter of importance Potassium iodide (KI) 0 3 to 0 6 Gm (5 to 10 grains) or Lugol s solution in 0 3 cc to 1 8 cc (5 to 30 minim) doses three times daily may be given In about 10 days marked improvement in the patient s condition will take place and this is reflected in the slowing of the pulse the reduction in the basal metabolic rate and improvement in all the symptoms of thyrotoxicosis Operation during this temporary period of induced calm can be performed with much less risk

Sedatives should be used during this preparatory regime to secure proper rest and thiamin chloride (vitamin $B_1$) should be given The best time for operation is determined by observing the pulse the basal metabolic rate and the weight in each individual Usually a period of 10 days is sufficient in uncomplicated cases

Digitalis should be given pre-operatively in every case of congestive failure regardless of the presence or absence of auricular fibrillation The amount of digitalis tolerated by a patient who has a greatly increased metabolic rate is of course much higher than can be administered safely to another patient of the same weight who has a normal basal metabolic rate

Auricular fibrillation is present in about 10 per cent of all patients suffering from thyrotoxicosis and in about 50 per cent of those who show signs of congestive failure The best treatment for this arrhythmia is sub total thyroidectomy Spontaneous return to normal rhythm following operation may be expected in a large percentage of cases

Congestive Failure In the pre-operative management of congestive failure the usual measures are employed (Chapter 2) Bed rest restriction of fluid intake relief of pressure caused by accumulation of fluid in serous cavities and the use of mercurial diuretics constitute the measures of greatest value The attempt should be made pre-operatively to control failure since operation in the presence of failure usually means subjecting the patient to an increased risk

If auricular fibrillation does not disappear spontaneously following operation quinidine should be given (page 383) The chances of restoring normal rhythm by the use of this drug are excellent if the usual rules for its administration are followed I do not think that it is of any value to establish quinidine maintenance in thyroid patients following operation after normal rhythm returns for the simple reason that the cause under lying the arrhythmia has been removed Recurrence of fibrillation is rare unless there is also a recurrence of the hyperthyroidism

Pre-operative control of the nervous symptoms is essential The almost specific effect of iodine in calming the pulse rate may likewise cause the

patient to be less apprehensive. To fortify its action occasional doses of phenobarbital or chloral hydrate may be used. The diet should be made up mainly of carbohydrates and should have a high caloric value because of the increased basal metabolic rate. Proteins should not be limited when the serum proteins are low and there is a tendency to edema.

The crisis or storm that may occur following thyroidectomy is caused by a severe toxic upheaval or an exaggerated hyperthyroid state. Within a few hours after the conclusion of the operation fever, tachycardia, restlessness, sweating, nausea or vomiting may appear. These symptoms call for prompt treatment. It is the common belief that this crisis develops as the result of flooding the system with the internal secretion of the thyroid gland at the time of operation. However, poor medical preparation of the patient will often predispose to this complication. If adequate attention is given to the cardiac condition and to the liver stores of glycogen before operation, serious storms may be avoided in most instances, although much depends on the judgment of the surgeon in choosing the proper time to operate. When the basal metabolism and the pulse rate reach their lowest level following iodine administration, the thyroid secretion is being held in the acini, and this is the time the surgeon chooses for operation —usually during the second week of pre-operative treatment. If the optimum time for operation is allowed to pass, toxic signs may appear in spite of treatment, and operation at this time increases the risk of crisis. The second preparation of the patient after omission of iodine for a time must be carried out, but is usually not so successful.

Surgical judgment is most important in the prevention of thyroid crisis. When the thyrotoxic state is poorly controlled by the medical regime, there will be less risk of this postoperative complication if a two-stage operation *is performed. The anesthetic and the anesthetist are* both important. Many surgeons prefer local anesthesia, but any light anesthesia that permits a liberal amount of oxygen to be available at all times may be used.

If a postoperative crisis appears, iodine should be given immediately. It is usually possible to administer a sufficient quantity by mouth, but in emergencies it may be given intravenously. The rising temperature should be controlled by ice bags or ice packs. Frequent temperature readings are necessary during the period of crisis. An oxygen tent is essential, but if it *is not available, the nasal catheter method may be used (page 98)*. Oxygen has been found to have a favorable influence on the temperature rise, the dyspnea and the cough, while the extreme restlessness of the patient should be controlled by sufficient morphine. Attention should be paid to the amount of fluids given, since the increase in the basal metabolic rate, the fever, the operation and the anesthetic place a considerable drain on the body stores. This need can be supplied together with an essential food substance by giving injections of 500 to 1000 cc of a 5 per cent glucose *solution as often as indicated. In some cases 5000 cc in 24* hours are required. At times reopening the operation wound in the neck and draining

out any serum present may swing the balance in a favorable direction during a postoperative crisis

**Irradiation** of the thyroid as a substitute for operation has been gaining in popularity and is the method of choice in cases where the risk of operation is too great It may occasionally bring about enough improvement to enable operation to be safely performed at a later date The same medical preparation of rest and iodine should be employed when treatment by irradiation is used These courses should be planned and given by a competent radiologist and the results controlled by frequent determinations of the basal metabolic rate

Roentgen ray therapy is worthy of consideration in the treatment of hyperthyroidism in children The disease is rare in children under 12 but a number of instances have been reported between the ages of 12 and 15 Since this period of life is important from the endocrine standpoint Rose and Pendergrass[3] suggest a trial of roentgen ray therapy in order to avoid the necessity of removal of the gland and report their results in 10 cases In five cases there was disappearance of the goiter and in eight cases recovery was complete The average dose was 1950 roentgen units over a period of six months If improvement is not observed in three months continued use of irradiation is unwarranted Should exacerbation of the disease occur at any time the treatment is stopped and the patient prepared for operation

## ILLUSTRATIVE CASES

### Hyperthyroidism—Death from Edema of the Larynx Following Thyroidectomy—Autopsy

**Case 57** Mrs J B a colored housewife of 24 was admitted to the Philadelphia General Hospital on 12/29/34 complaining of loss of weight nervousness and palpitation The symptoms had been present and progressive for a year

Physical Examination B P 140/80 There was a diffuse enlargement of the thyroid exophthalmos and tremor A systolic apical murmur was heard and the first sound over the same area was accentuated There was no increase in cardiac size

Laboratory Data Basal metabolic rate plus 39 per cent The electrocardiogram showed sinus tachycardia The roentgen examination showed an overactive heart but no enlargement The blood and urine were negative

Subsequent Course Following rather prolonged preparation a subtotal thyroidectomy was performed on 1/24/35 The patient left the operating room in good condition but an hour later developed stridor due to edema of the larynx and died in spite of emergency tracheotomy

Autopsy (Fig 137) The heart was of normal size and consistency but showed acute dilatation of the right ventricle No lesions were present

**Discussion** The development of stridor following thyroidectomy suggests either injury to the recurrent laryngeal nerves or as shown in this case edema of the larynx Hemorrhage into the operative field with pressure on the trachea by a hematoma may cause the symptom Examination of the larynx should be done at once to determine the cause and if relief

is not promptly obtained especially if cyanosis develops an immediate tracheotomy is indicated

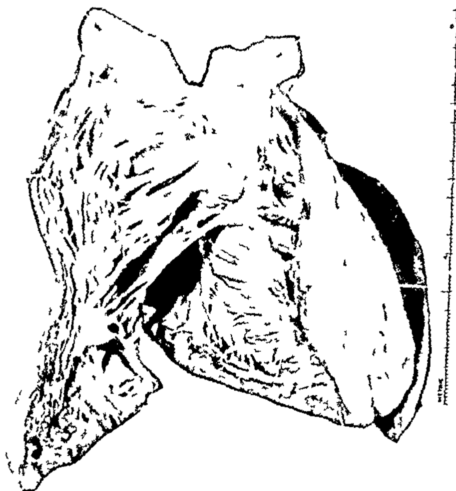


FIG 137 The heart in hyperthyroidism Note absence of hypertrophy and the delicate heart valves (Autopsy No 28 782 Philadelphia General Hospital)

The heart in this case is typical of uncomplicated thyrotoxicosis Note the relative increase in the size of the right ventricle and the normal, delicate heart valves

THYROTOXICOSIS COMPLICATED BY PAROXYSMS OF AURICULAR FLUTTER—COMPLETE RELIEF FOLLOWING SUBTOTAL THYROIDECTOMY

**Case 58** Mrs S B a housewife of 56 was first seen March 1 1936 complaining of loss of weight nervousness and spells of rapid heart action

HISTORY All symptoms began following a series of domestic difficulties six months before the initial examination During this period there was a weight loss of 30 pound

The patient had previously been treated for a nervous breakdown' When spells of rapid heart action and increase in blood pressure were noted she was diagnosed hyper tensive heart disease and given digitalis in large doses This had no effect She was unable to sleep because of h r nervousness and rapid heart Slight dyspnea was present No edema

PHYSICAL EXAMINATION B P 170/80 Pulse 140 Patient emaciated Skin was warm and moi t much more so than would be expected in a person of her age There was

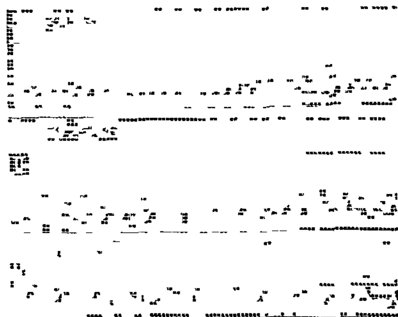


FIG 138 The elect ocardiogram (lead 2) taken on 3/17/36 during one of attacks shows presence of aur cular flutte There are varying degrees of ventricular response A portion of the sam l d of a trac ng tak n eight days later show s sinus tachycard a Note high pointed P av s A record taken at t me of follow up visit eight months fter operat on shows s nus rhythm ate 80 and normal P waves

tremor and sl ght thyroid swell ng No exophthalmos There were pulsat ons noted in the neck and a d ffu apex beat was seen in the m d clavicular l ne There was no card ac enlargement o pe cuss on A syst lic murmur was heard over the mitral area

LABORATORY DATA B M R plus 49 per cent Fluoroscopy showed characteristic ove ct on The as no ncrea e in cardiac size

Th el ctro a diogram (F g 138A) m de at the time of the first examinat on (3/17/36) during a paroxysm of rap d heart act on showed that the mechanism was auricular flutte Th e was a vary ng ventricular re ponse On 3/ 5/36 s nus tachycardia as present Note th ncrease in height of the P waves in the tracing (Lead 2) On 1?/1 / f th lect card ogram w as normal

CLINICAL DIAGNO IS A Et olog c Hyperthyro dism B Anatomic No ca diac enlarge ment C Phy iologic Paroxysmal aur cular flutter D Functional Class fication Class 1 Ther peut c Classificat on Class D

DISCUSSION Three weeks later (one week to convince two weeks to prepare) a subtotal thyroidectomy was performed A very small nodule of

thyroid tissue was *allowed to remain on each side of the trachea* The convalescence was smooth The paroxysms of auricular flutter did not recur following operation

On discharge from the hospital *on the eighth postoperative day* the basal metabolic rate was plus 29 per cent the pulse 100 and normal sinus rhythm was present Potassium iodide was continued in 0 3 Gm (five grains) doses after meals The *patient gained three* pounds during the first postoperative week

Six weeks later the basal metabolic rate was plus 10 The pulse had dropped to 90 and there was considerable subjective *improvement* B P 130 90 No cardiac symptoms were present A total gain in weight of 15 pounds since the operation was recorded at this time

Follow up examination three months after operation showed a basal metabolic rate of minus four per cent The weight was now 130 pound a further increase of 15 pounds since the last visit No cardiac symptoms Pulse 80 B P 140 80

When this patient was first seen she was an invalid confined to bed nervous emaciated and discouraged A diagnosis of hypertensive *heart* disease had been made and her outlook was considered poor The importance of making the correct etiologic diagnosis in heart disease can be *appreciated* when we view the patient *15 weeks later* active *quiet and* plump restored to health and able to carry on the usual activities of a woman of her years

*This result teaches us never to overlook the possibility of thyrotoxicosis* in elderly patients who do not present the usual eye signs or thyroid tumor particularly when these patients have increased blood pressure a history of loss of *weight nervousness and spells of rapid heart* action Here is a curable type of heart disease

### Exophthalmic Goiter—Subtotal Thyroidectomy Followed by Postoperative Hypothyroidism

**Case 59** Mrs G P *an Italian house ife of 7 as first seen in* October 1935 complaining of nervousness and palpitation All the classical signs of thyrotoxico is were present on physical examination *The basal metabolic rate was plus 60*

Preparation for operation extended over two weeks and consisted of the usual measures. At the end of this time *the* basal metabolic rate was *plus 35 per cent and the nervous* symptoms showed considerable improvement There had been a two pound gain in weight

A subtotal thyroidectomy was performed and the convalescence was *uneventful Three* months after discharge the patient moved to another city and was lost sight of for three years On November 1938 she returned for study with a diagnosis of chronic nephritis and secondary anemia There was swelling of the face hand and legs B P 1 0/80 The heart was slightly enlarged The urine showed a trace of albumin specific gravity 1 0 0 but no casts The basal metabolic rate was minus 40 per cent and the blood cholesterol 285 mg per 100 cc

*Discussion* The *diagnosis* of nephritis *in this instance* was made without a full consideration of the past medical history and was based on insufficient evidence The color the edema and the slight trace of albumin were secondary to the postoperative hypothyroidism and not to a renal lesion

The surgeon ordinarily leaves behind a remnant of thyroid tissue the

size of which is governed by his judgment as to the nature of the disturbance evident in the resected gland This glandular substance that remains may support the bodily needs for a time and then burn out and become entirely inactive Consequently a hypothyroid state develops

In some patients who do not complain of any symptoms a postoperative basal metabolic rate a few points below normal is not an indication for thyroid administration However where actual symptoms of myxedema develop treatment with thyroid gland is indicated

This girl is certainly far better off in her present condition than she was when her life was threatened by the hyperthyroidism This fact must be pointed out at the start and unjust criticism directed toward the surgeon should not be allowed The patient should be thankful since this evidence points to final victory over her thyrotoxic state Myxedema is easily managed (page 368)

## Toxic Goiter—Death Following Postoperative Thyrotoxic Crisis—Autopsy

**Case 60** M B a colored female of 48 was admitted to the Philadelphia General Hospital with a chief complaint of nervousness This symptom was noted a year before and was accompanied by swelling of the neck Following this she developed tremor excessive sweating dyspnea tachycardia and a weight loss of 0 pounds Continuous iodine therapy (five drops t i d ) for the past five months gave some relief

Physical Examination B P 190/90 Exophthalmos thyroid enlargement tremor tachycardia Heart rate 150 rhythm regular apex beat diffuse and wavy in the mid clavicular line The first sound was accentuated at the apex There was a systolic apical murmur A moderate sclerosis of the vessels was observed

Laboratory Data B M R plus 96 to plus 39 Roentgenogram heart overactive but not enlarged Substernal thyroid

Blood count Hemoglobin 8 per cent (Sahli) Red blood cells—4 700 000 White blood cells—6 000 Polymorphonuclears—47 per cent lymphocytes—52 per cent basophiles—1 per cent

Procedure Bed rest iodine sedatives glucose for two weeks When the basal metabolic rate fell to plus 39 per cent and symptoms subsided a subtotal thyroidectomy was performed

Outcome On the first postoperative day the patient was restless and irrational The pulse rate mounted steadily to 170 180 and the temperature to 106 F There was dyspnea Death occurred on the second postoperative day in spite of the usual restorative measures

Autopsy Heart and lungs were removed en masse and are shown in Fig 139 Note the striking prominence of the pulmonary conus and the pulmonary artery The latter was elongated and widened its intrapericardial portion measuring 6 cm in length and the diameter at the base three cm It overshadowed in size the aorta which was likewise somewhat elongated The heart was normal in size with a muscle of firm consistency The valves were normal

Discussion Preparation in this case was carried out along the usual lines The postoperative storm that was directly responsible for the death of the patient might have been prevented if a longer period of preparation had been allowed A multiple stage operation might also have averted the postoperative crisis However storms may occur after partial removal or ligation as well as following subtotal thyroidectomy

Iodine is the main drug at the time of postoperative crisis (page 358) The routine use of iodine pre-operatively has removed the main objection

to one stage total thyroidectomy and the risk as shown by figures from many clinics has decreased to le s than 1 per cent

Some observers claim that the discharge of adrenalin accounts for the postoperative storm In this re pect we can realize how important it is

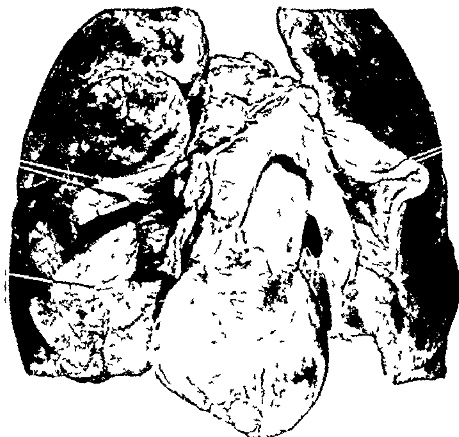


FIG 139 The heart in hyperthyroidism Note th enlargement of the pulmonary artery pulmonary conus and the right ventricle (Autopsy No 9 530 Phila l lphia General Hospital)

to save the patient all we can from exposure to disturbing situations before during and following operation Care should likewise be used in prescribing adrenalin for these patients no matter what the situation may be

RHEUMATIC HEART DISEASE WITH SUPERIMPOSED THYROTOXICOSIS—SUDDEN DEATH ON FIFTH POSTOPERATIVE DAY—AUTOPSY

**Case 61** Mrs C H an American house wife of 45 was adm tted to the Woman s College Hospital on 9/ 1/34 complaining of palpitation nervousness and weakness of two months duration

HISTORY The patient had rheumatic heart disease for some years that required little treatment until two months before admission At this time she noticed increasing nervousness, tremor weakness and palpitation Her dyspnea increased and there was slight edema of the ankles in the evening one month before admission During two months she lost 18 pounds in weight

PHYSICAL EXAMINATION BP 150 90 Heart rate 120 rhythm totally irregular Tremor Skin warm and moist There was a slight swelling of the right lobe of the thyroid gland The apex beat was palpated in the sixth interspace cm outside the mid clavicular line There was an accentuation of the first sound and a diastolic murmur at the apex The basal metabolic rate was plus 4 Th rest of the examination was irrelevant

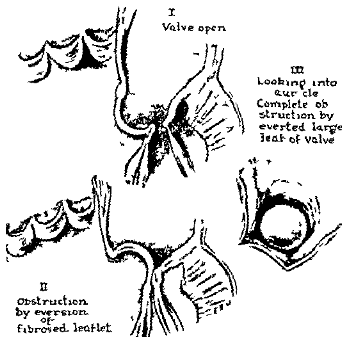



FIG 140 Evers on of the mitral l aflet

IMPRESSION A Etiolog Rh u atic Inactive Thyrotox cosis B Anatom c Cardiac enlargement M t al steno s C Physiologic Au icular fibr llation D Functional Classification Cla 3 Th ap ut Cla s fication Class D

PROGRESS On Lu ol s lut n the pulse rate decreased to 80 but emained ir egular The pat ent was improved N gns of congest ve failure were evid nt A subtotal thy o d ct my was p rformed on th th teenth hospital day

The pat nts course dur ng the fir t five postoperative days was tormy She was ir ational unt l th th d day On the sixth day because of the continuation of auricular fib illation h was g ven a mall test dose of qu n d ne sulfate 0 2 Gn (3 gra ns) The next day ho tly aft r a second dose of 0 3 Gm (5 gra ns) was given she d ed sudd nly

AUTOPSY H art weight 330 Gm The muscle was pale and soft The left vent icle wa dilat d Th r ght v nt cle as considerably dilated and hypertrophied The l ft aur cl was normously dilated with th ckened and scarred endocardium especially on

the posterior wall On the anterior mitral leaflet there was a thickened fibrotic area This mass of tissue bulged up into the auricle and pressed against the posterior leaflet completely blocking the stenotic mitral orifice (Fig 140) The aortic valve leaflets were thickened puckered and basically incompetent The pulmonary artery was dilated and atheromatous

**Discussion** This patient s death can be attributed to sudden (forward) failure of the circulation resulting from an eversion of the mitral leaflet The cause of this unusual accident remains a mystery Since it followed an attempt to restore normal rhythm by quinidine the situation may be discussed from this angle

It is the usual practice to administer quinidine postoperatively in all cases where auricular fibrillation does not cease spontaneously By the fifth postoperative day in over 60 per cent of the cases normal rhythm will replace the fibrillation This patient was given the usual test dose The next day a 3 grain capsule was administered and shortly before the sudden death of the patient the intern had reported the pulse to be regular Sudden death after the use of quinidine in patients with fibrillation of long standing may follow the dislodging of a clot in the auricular appendix with the production of an embolus Consequently the drug should never be used in these cases particularly if there is evidence of congestive failure

In this patient the fibrillation was recent and was brought about by the added burden of the thyrotoxicosis However advanced rheumatic heart disease with mitral stenosis complicated the picture The beginning of coordinated auricular contractions may have created sufficient change in intracardiac pressure to cause the accident The exact mechanism of this rare happening has so far been unexplained

## HYPOTHYROIDISM

Hypothyroidism or myxedema results from a deficiency in the thyroid hormone and is likewise attended by a variety of cardiac manifestations This state may develop in early childhood or in adult life and its effect on the organism in each instance will be quite different Means [90] divides hypothyroid states into (1) infantile myxedema or cretinism (2) childhood or juvenile myxedema and (3) adult myxedema or Gull s disease

### Etiology

Sporadic cretinism follows thyroid atrophy but the exact cause of this change is unknown The cretin shows retardation of growth and development associated with delay in bone ossification epiphyseal union and dentition The face is quite characteristic with its stupid expression thick lips and protruding tongue For this reason cretins resemble one another

**Juvenile myxedema** produced when there is lack of thyroid secretion prior to puberty occupies a place between cretinism and myxedema It differs from cretinism since normal or nearly normal amounts of thyroid

secretion are available for the early years of growth and development although the supply is cut off before the process has been completed

**The adult type** of myxedema is a rare disease caused by atrophy of the thyroid gland that develops spontaneously or as the result of a previous inflammatory process This manifestation of hypothyroidism is particularly interesting to us because of the profound circulatory alterations that accompany it The generalized infiltration of skin with a mucus like substance that does not pit on pressure characterizes the condition (see Fig 142A) The myxedema heart is often uniformly enlarged owing to infiltration with the same material *Skeletal muscle shows on examination a pale* edematous appearance and fluid may collect sometimes in considerable quantities in the serous cavities [9]

## Signs

In myxedema the reduction in the basal metabolic rate is striking Levels of minus 40 and below are not uncommon This decrease is accompanied by profound alterations in the protein metabolism with retention of protein in the intercellular spaces and increase in the proteins and cholesterol of the blood The pulse rate drops the minute volume output decreases and the whole picture is reflected fluoroscopically in the lazy dilated heart of the myxedematous patient The activity of the organism drops very close to the level observed in hibernating animals

## Course

**The onset** of the myxedematous state is slow and marked progress usually takes place before it is detected clinically This may be caused by the gradual destruction of thyroid tissue by some unknown process which may start years before the patient appears for treatment Its nature therefore generally eludes detection

## Symptoms

Symptoms develop in proportion to the reduction in the metabolic rate When this is of slight degree only mild symptoms appear Sweating decreases and the patient ceases to complain of the summer heat * and since the body fires are burning low there is a dislike for the cold weather The myxedematous patient is contented to sit down and take little interest in the passing scene Mental dulness increases and considerable time is consumed by sleep Constipation is complained of and headaches usually appear The hair becomes thin particularly along the back of the neck and deafness may be noted

**The cardiac symptoms** are part of this general picture Dyspnea is first to appear and soon becomes marked on slight exertion Some patients develop cardiac pain that is relieved by the administration of thyroid gland however as Means points out [6f] it is far more common to have thyroid therapy produce angina than relieve it The heart is enlarged the rhythm

* A very suggestive symptom in Philadelphia

regular the rate slow and the sounds are usually feeble If another type of heart disease is already present a superimposed myxedematous state may precipitate congestive failure In spite of these numerous characteristic symptoms it is surprising how often the diagnosis of myxedema is missed

TREATMENT

**Thyroid Medication** The effect of thyroid therapy in myxedema is most gratifying since it restores to normal all bodily processes Tablets of dried thyroid gland are efficient and less expensive than other preparations Means has found after a careful study of 50 cases that requirements show little variation A dose of one half of a grain a day of dried gland should keep the basal metabolic rate of a myxedematous subject at a level of minus 20 One grain daily will maintain it at minus 10 one and one half grains at approximately minus 5 and three grains daily at a normal level IT IS OF THE GREATEST IMPORTANCE THAT THE MYXEDEMATOUS STATE SHOULD BE CONTROLLED ON THE SMALLEST DOSES OF THYROID COMPATIBLE WITH A NORMAL EXISTENCE Inasmuch as there is no way of knowing the basal metabolic rate before the onset of myxedema the patient must be regulated by clinical trial and the effect of thyroid therapy on the symptoms closely studied The minimum dosage that gives the desired effect is then continued for the rest of the patient s life

In prescribing or dispensing thyroid tablets the physician should remember that there are differences in the tablets supplied by the various manufacturers and that a patient balanced satisfactorily on one brand may show symptoms of hyperthyroidism on another The figures given above refer to U S P thyroid Variations in the strength of the different preparations will usually be indicated on the label so the necessary adjustments can be made It is however unwise to change the brand of thyroid used Thyroid gland U S P should be prescribed because of its lower cost and the uniformity of its action Familiarity with the dose of this standard preparation is all that is necessary The addition of 1 cc (16 minims) of dilute hydrochloric acid taken with meals may enhance the action of the thyroid gland in the presence of gastric hypo acidity

When thyroid medication is begun there is a latent period of from six to eight days before its action is evident An elevation in the basal metabolic rate first appears This is produced by an increase of fat and carbohydrate metabolism The blood sugar is increased as the glycogen store of the liver is depleted At the same time sugar tolerance is diminished and glycosuria may appear In the usual subject these changes may result in a loss of weight If the administration of the thyroid substance is continued restlessness irritability, sweating tachycardia and muscle tremors will be produced

THYROXIN is no more effective than dried thyroid gland This preparation can be administered intravenously but the indication seldom arises The action of thyroid gland by mouth is just as satisfactory and the usual risks that attend any form of intravenous therapy are avoided Thyroxin

administered by mouth is absorbed less uniformly than dried thyroid and consequently for routine use aside from the greater cost it does not display the efficient action of the latter preparation

SEQUELAE Under treatment with thyroid the size of the myxedema heart shrinks the greatest reduction occurring in the largest hearts This is strong proof that the condition is not one of true hypertrophy but rather a manifestation of the myxedematous state Heart size returns to normal slowly as the fluid is eliminated and the tone improves If this does not occur we should search for the presence of another complicating type of heart disease

Care must always be exercised in prescribing thyroid for myxedematous patients for too enthusiastic a start often precipitates grave symptoms Since myxedema favors the development of arteriosclerosis it is not surprising that anginal attacks occur when the basal metabolic rate and consequently the cardiac work are increased to too great an extent in the beginning of the treatment If angina appears the thyroid medication should be temporarily discontinued and when begun again smaller doses should be prescribed

Digitalis has no effect in a case of true myxedema in fact it is poorly tolerated The only indication for its use is the congestive cardiac failure which often occurs when myxedema complicates some other type of heart disease

Some degree of anemia accompanies myxedema and generally can be attributed to the effect of the disease on the blood forming organs When of extreme degree it may accentuate the cardiac symptoms particularly the dyspnea Improvement is then speeded by giving ferrous sulfate 0 6 Gm (10 grains) t i d with the thyroid tablets

## ILLUSTRATIVE CASES

### HYPOTHYROIDISM FOLLOWING IRRADIATION—DEATH FROM CORONARY DISEASE—AUTOPSY

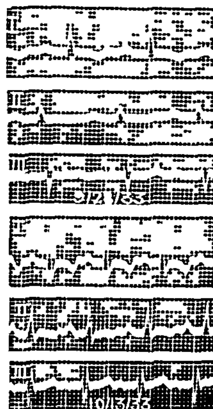
**Case 62** Mrs E K a white housewife of 43 gave a history of hyperthyroidism two years prior to admission to the Philadelphia General Hospital on 8/ 4/33 She received roentgen ray treatment over this entire period Although the nervousness and palpitation were much improved during the last six months she had noticed weakness, dyspnea dryness of the skin loss of hair and swelling of face and legs The skin became thickened and the nail brittle

PHYSICAL EXAMINATION Exophthalmos and widening of the palpebral fissures were present The thyroid was not palpable Rales were present in both lung bases A moderate bilateral pleural effusion was found The heart rate was slow and the sound distant The borders could not be satisfactorily determined because of pleural fluid

LABORATORY DATA Urine showed a cloud of albumin specific gravity 1 010 Blood Hemoglobin 70 per cent (Sahli) Red blood cells 2 800 000 White blood cells 9 000 polymorphonuclears 53 per cent lymphocytes 47 per cent Kahn negative Cholesterol 385 Basal metabolic rate minus 30 per cent. The first electrocardiogram showed low voltage of QRS prolongation of the P R intervals to 0 8 seconds and a left axis deviation Two weeks later (Fig 141A) marked alterations in the T waves appeared and a Q wave in lead 3 was noted

PROGRESS The patient showed very little response to bed rest, diuretics and cautious thyroid administration and died suddenly six weeks later

AUTOPSY Thyroid Considerable fibrosis was present and no thyroid tissue was visible in the section The heart showed hypertrophy of all chambers There was considerable sclerotic thickening of the mitral and aortic valves and extensive atheroma of the aorta



A



B

FIG 141 A The electrocardiogram on admission Note bradycardia the low voltage of all waves and the flat T waves A record taken two weeks later following administration of thyroid shows increased voltage The T wave changes were probably caused by alterations in the blood flow to the heart muscle B Atheroma of the aorta associated with hypothyroidism (Autopsy No 26796 Philadelphia General Hospital)

DISCUSSION Myxedema in the adult may be spontaneous or induced The type that follows irradiation or surgical removal of all thyroid tissue (cachexia strumipriva) is much more common today The roentgen ray affects all thyroid cells and if the treatments are continued over a long period may lead to a total suppression of the internal secretion This patient had typical hyperthyroidism two years before admission An excellent result was obtained at the start with irradiation and the treat

ment was continued over a longer period than was necessary with the development of hypothyroidism and later a frank myxedema

High blood cholesterol values are usually obtained in myxedema, and these may be seen to decrease after the administration of thyroid extract The level of the blood cholesterol and the basal metabolic rate vary inversely In non toxic goiter the blood cholesterol is normal while in thyrotoxicosis and thyroid crisis it is low Consequently blood cholesterol determinations are valuable in diagnosis and in following the progress of treatment

This patient s early death from coronary disease the advanced degree of atheroma discovered at autopsy and the high blood-cholesterol value that was associated with her myxedematous state are more than coincidental When the dangers of thyrotoxicosis were avoided she developed the high blood cholesterol which probably speeded up a sclerotic process already present Blumgart has been unable to confirm this view and reports no evidence of rapidly progressing arteriosclerosis among the patients subjected to total thyroid ablation for relief of cardiac pain and congestive heart failure[34]

### Myxedema Complicated by Coronary Arteriosclerosis—Death Following Second Attack of Coronary Thrombosis

**Case 63** N T a bank clerk of 55 was first seen with his physician on November 10 1939 At this time he showed all the typical signs and symptoms of myxedema Two weeks before he had experienced a sudden attack of precordial pain in the middle of the night this persisted for some hours and required a hypodermic injection of morphine for relief

Physical Examination B P 140/80 Pulse 55 There were noted a puffy face sparse eyebrows dry skin and thin hair (Fig 14 A) The heart sounds were distant and soft systolic murmurs were heard over the apex and aortic area The rhythm was regular The heart was not enlarged to percussion

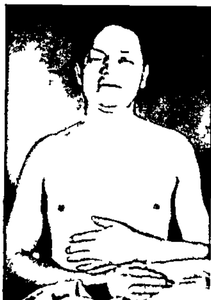
Laboratory Data The electrocardiogram showed low voltage and in addition changes that suggested recent coronary occlusion (Fig 14 C) The roentgenogram showed slight cardiac enlargement (Fig 14 B) The blood cholesterol was 370 mg The blood count was normal urine and Wassermann negative The basal metabolic rate was minus 4

Clinical Diagnosis A Etiologic Myxedema B Anatomic Coronary sclerosis Coronary occlusion Cardiac infarction C Physiologic Sinus bradycardia D Functional Classification Class 4 Therapeutic Classification Class E

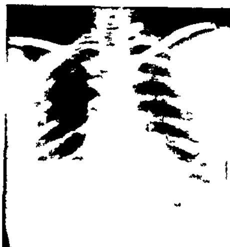
Discussion Means[66] believes that the occurrence of coronary thrombosis in myxedema is largely an accidental association He states that persons who develop myxedema usually do so in the age periods when coronary thrombosis is also common and he is not surprised that certain patients should have both diseases

Some of the patients with myxedema have angina before thyroid treatment is begun Others are free of pain until thyroid medication is pushed too fast for the caliber of the coronaries and then seizures develop The treatment in these cases consists in balancing the amount of daily thyroid ration to give the greatest relief from the symptoms of myxedema without aggravating the angina Many times this dose will be under one

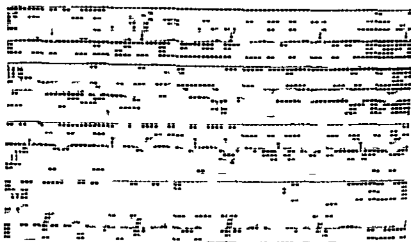
grain of thyroid gland a day Nitroglycerine should be given as often as needed to control the angina



A



B



C

FIG 142 A Myxedema B Roentgenogram shows slight cardiac enlargement C The electrocardiogram in myxedema Note the low voltage of all waves The S T intervals in leads 2 and 3 show doming that is quite suggestive of old posterior coronary occlusion Lead 4F is normal except for low voltage

The association of arteriosclerosis with myxedema cannot fail to call to mind the relationship between diabetes and similar changes in the

coronary tree In both diabetes and myxedema arteriosclerosis is an early complication and acute coronary occlusion often terminates the picture in each instance Joslin believes that the problem of arteriosclerosis in diabetes will be solved if complete oxidation of fat is accomplished High cholesterol values are found constantly in diabetes as well as in myxedema With such a large cholesterol supply available in the blood stream it is natural that investigators should conclude that this substance finds its way into the vessel walls and becomes concentrated in the spots where necrosis develops Further support is given to this view by laboratory workers who have fed diets high in cholesterol to animals with the production of similar lesions

Whether or not we make use of this experimental data in the management of our diabetic and myxedematous patients we must admit that many problems (and secrets) physiologic biologic and pathologic are closely linked to that fascinating chemical compound, cholesterol

# 12

# CARDIAC ARRHYTHMIAS

Intelligent pulse palpation is an invaluable short cut to a great deal of information essential to the management of the cardiac patient. Moreover, there are important inferences which the pulse and only the pulse enable us to draw. While the rate, size and compressibility of the pulse are important notations, rhythm deserves the most careful appraisal. Attention to a few fundamental rules and constant practice should enable every physician to diagnose the majority of the arrhythmias clinically, especially when pulse palpation is combined with careful auscultation of the heart. Needless to say, this is a great advantage in emergencies when the electrocardiograph is not available.

We can no longer state that the rhythm of the heart is "irregular" or "slightly irregular." The type of the arrhythmia should be put down, for it can be determined with very little difficulty by the general practitioner at the bedside. In fact, much of our present knowledge of the cardiac irregularities is a heritage from a country doctor, since the greatest part of the contributions of Sir James Mackenzie were made while he was in general practice at Burnley. Instruments of precision were perfected by him to test his theories, but when the many problems surrounding the arrhythmias were solved, he discarded the instruments and returned to continue his investigations at the bedside of the patient.

Irregularities in the rhythm are present many times in the absence of heart disease. However, the impression they make on the nervous system of the patient gives them a place of importance when treatment is planned. The patient whose nervous system is receptive to the stimuli of these abnormal beats is usually the patient who is constantly engaged in the palpation of the radial pulse. Consequently, a harmless arrhythmia may be the starting point of a neurosis that eventually leads to as much ill health as an organic lesion.

A clear understanding of the arrhythmias comes with a thorough knowledge of the physiology of the cardiac mechanism (Chapter 24). *Abnormalities of the cardiac action arise when the rate of impulse production is increased or decreased, when the impulse has its origin outside the regular sinus node or when disease processes block the normal distribution of the impulse.* Before considering the treatment of the cardiac irregularities separately, a few general rules that may prove useful in their recognition will be mentioned.

## DIAGNOSIS

**Age Relations** If an arrhythmia is present in a child under eight years of age it is almost certainly sinus arrhythmia Heart block caused by a congenital defect in the septum may be noted at birth but it is rare (page 343) Auricular fibrillation is seldom present to complicate rheumatic or congenital lesions in the very young unless cardiac failure is imminent (page 348)

**The cardiac rate** is an important consideration in the diagnosis of any arrhythmia Persisting and regular ventricular rates below 40 point to complete heart block When a pulse rate over 140 suddenly appears in a patient at rest and continues at this level or higher for a space of hours to return suddenly to normal a paroxysm of tachycardia or flutter is usually the mechanism Pressure over the carotid sinus in the neck serves to differentiate since it causes marked slowing in the ventricular rate if flutter is present while if it has any effect at all on a paroxysm of tachycardia it will stop it at once and the pulse rate returns suddenly to normal

**Premature Beats vs Auricular Fibrillation** If the heart's rhythm is irregular with a slow ventricular rate it will require careful auscultation to differentiate between frequently recurring premature beats and auricular fibrillation If the patient is exercised or given an inhalation of amyl nitrite to speed up the pulse the differentiation is easy With auricular fibrillation the irregularity of the heart becomes more pronounced while in the presence of premature contractions the rhythm becomes rapid and entirely regular It is unusual for premature beats to persist with pulse rates above 110

**Alternation of the pulse** is a very important type of arrhythmia to diagnose because of the bearing it usually has on prognosis Pulse palpation may suggest its presence but it may be more readily demonstrated by using the sphygmomanometer (page 409)

**A skipped or dropped beat** at the wrist may be caused by premature contractions heart block or sinus arrest The last two are rare By listening over the precordium while palpating the carotid pulse the sound of the premature beat may be detected while precordial auscultation in patients who have heart block or sinus arrest reveals no extra sound during the pause An electrocardiographic examination may be necessary to distinguish between heart block with dropped beats and sinus arrest although in the presence of the former various signs that point to cardiac disease viz enlargement of the heart fever of a rheumatic process etc may be evident Sinus arrest is more likely to occur in healthy hearts while heart block never does

**Bundle branch block** may be suspected when a splitting of the first heart sound occurs in the presence of other evidences of cardiac disease

It is usually caused in these instances by asynchronous contraction of the ventricles

**The symptoms** that accompany the cardiac arrhythmias vary from the nervousness of the patient who experiences occasional premature beats to the coma and convulsions that are often seen in patients suffering from varying degrees of A V heart block The same irregularity in one person may cause no symptoms at all in the second Even marked disturbances of cardiac rhythm such as auricular fibrillation may be unappreciated by the nervous systems of some individuals while infrequent premature beats in susceptible patients may be accurately catalogued as to number and time of day of their occurrence

In all cases it is important to secure a detailed description of the type of irregularity causing the disturbance Many patients will give a description so characteristic that a diagnosis can be made on the history The sensations described as *skippings pulsations heart turning over* '*chokings* are nearly always due to premature beats Sudden racings and flutterings are terms generally used to refer to paroxysms of rapid heart action

Occasionally the sudden onset of a paroxysm of tachycardia may prove more than the myocardium of an elderly patient can stand in which event the symptoms of congestive cardiac failure may quickly appear The situation precipitated by the arrhythmia then assumes the nature of a medical emergency

Regardless of the type of irregularity complained of the final judgment in each case should rest on the result of complete physical examination Particular attention should be given to the blood pressure and the attempt should be made to demonstrate the presence of pulsus alternans if the clinical findings warrant the search The jugular bulb should receive close attention and study Many times the waves observed here reflect a type of auricular activity that permits definite conclusions to be made in regard to the nature of the arrhythmia Careful precordial auscultation should be made during pulse palpation Carotid sinus pressure should always be practiced and its effect on the irregularity noted A careful evaluation of all the signs and symptoms of cardiac disease is always the deciding factor in estimating the importance of any arrhythmia and in planning management

## SINUS MECHANISMS

### Sinus Tachycardia

**Case 64** M R a physician of 9 came to the hospital for a cardiac study because of tachycardia of three weeks duration The average rate was 110 onset and offset were gradual

Physical Examination B P 108/70 The heart was not enlarged No murmurs were heard The electrocardiogram that the patient requested revealed only a sinus tachycardia with a rate of 1 0 Further study however showed a temperature of 99 3 F and an active tuberculous lesion at the right apex of the lung

Discussion The heart many times gives warning of trouble in a neighboring organ This case history shows how dangerous it is to confine an examination to one region or organ of the body The sinus tachycardia here interpreted as heart disease was the result of the toxemia and fever of an active tuberculosis

Sinus tachycardia is likewise commonly produced by overindulgence in tea coffee tobacco and alcohol gastro-intestinal tract disturbances hemorrhage shock hyperthyroidism and cardiac failure The tachycardia of the latter condition is controlled by the regime described for congestive failure

## Sinus Arrest

**Case 65** Miss E J a student of 17 was referred to the Cardiac Clinic of the Woman s College Hospital by a school physician because of a slow irregular pulse Rate 48 Occasionally dropped beats were palpated at the wrist No precordial sound was heard on auscultation during the pauses Cardiovascular symptoms were absent and the past history was negative for rheumatic infection The physical examination except for the slow pulse and arrhythmia was entirely negative

Clinical Diagnosis Possible heart disease (patient with signs referable to the heart but in whom diagnosis of cardiac disease is uncertain) Functional Classification Class I Therapeutic Classification Class A Electrocardiogram needed to establish the diagnosis

Discussion The electrocardiogram showed the presence of sinus arrest (See Fig 180A) This girl was active in all forms of athletic activity and at the time of the examination was engaged in a school hockey tournament It is not uncommon for well trained athletes to show these manifestations of an increase in vagal tone consequently in this patient the bradycardia and sinus arrest were disregarded The latter disappeared when tincture of belladonna was administered in full dosage over the course of one week No treatment of course was indicated A follow up examination in three months showed that the sinus arrest had disappeared

## Sinus Arrest During the Course of Arteriosclerotic Heart Disease

**Case 66** R S a retired policeman of 67 was admitted to the Woman s College Hospital on 1/3/31 complaining of a sudden severe chest pain The pulse was 40 and the rhythm irregular

Physical Examination B P 100/70 T 99 2 Pallor and sweating were observed Circulatory balance was quickly gained but the pulse remained slow and irregular No precordial friction rub appeared

Laboratory Data The orthodiagram was normal Blood count normal Urinalysis negative Blood Wassermann negative

Electrocardiogram (see Fig 180B) sinus arrest.

Clinical Diagnosis A Etiologic Arteriosclerosis B Anatomic Coronary sclerosis Coronary occlusion (?) C Physiologic Sinus arrest D Functional Classification Class 4 Therapeutic Classification Class E

Discussion The slow pulse accompanying the sudden onset of chest pain suggested at first a posterior coronary occlusion but an electrocardiogram showed the presence of sinus arrest instead of the expected complete A V block This suggested that the artery supplying the sinus node was

the site of the lesion No signs of circulatory insufficiency attended the slow pulse rate and the treatment of this patient was the treatment of the coronary disease The rhythm returned to normal in one week and no irregularities were noted during the remainder of the patient s period of convalescence

This patient was seen again five years later when he was admitted because of a urinary tract obstruction secondary to prostatic hypertrophy and cardiac studies were repeated The sinus arrest was no longer in evidence but there was prolongation of the P R intervals to 0 24 second and a small Q wave in lead I The orthodiagram again showed no cardiac enlargement Routine physical examination on the second visit revealed a large pulsating abdominal mass proved by roentgen study to be an aneurysm of the abdominal aorta In spite of these evidences of wide spread sclerosis a prostatectomy was successfully performed and convalescence was smooth At a follow up examination two years after the prostatectomy the patient was ambulatory and his only complaint was a mild angina on effort

Sinus arrest in Case 65 was allied to vagal stimulation and sinus arrhythmia in a healthy heart and we may assume that it was a transient functional alteration However in Case 66 the disorder is most likely associated with a deficiency in the coronary circulation Since some degree of A V block was also present there were likewise some circulatory changes in the region of the bundle The vertigo complained of by the patient on his first admission was probably caused by the cerebral anemia that followed the sinus arrest The occasional dropping of a single beat will rarely produce any subjective disturbance other than palpitation in patients who are sensitive to alterations in their cardiac action In occasional cases the blocking in succession of two three or more beats may cause symptoms of faintness or even syncope to appear and the resemblance to Adams Stokes attacks will be very close In patients who complain of occasional fainting spells in the absence of signs of disease in the nervous system or heart sinus arrest must be considered as a possibility

### Sinus Bradycardia Caused by Hypersensitive Carotid Sinus

**Case 67** R F a male executive of 50 was admitted to the Woman s College Hospital on 7/15 36 for study because of vertigo and faintness of a year s duration Two attacks of syncope occurred while at work the week before admission The periods of unconsciousness were brief neither exceeding two minutes, and there were no convulsive movements Each attack was attended by an extremely slow radial pulse (50 to 60) The physical examination of the chest was entirely negative An orthodiagram showed a heart well within the limits of normal in size and shape The electrocardiogram (see Fig 196) showed bradycardia and wandering pacemaker A G I tract examination showed a moderate degree of visceroptosis

**Discussion** A marked sinus bradycardia was present in this case associated with obesity and hypotension Pressure over the carotid sinus on the right slowed the heart rate and caused faintness In 1927 Hering showed that this phenomenon was caused by a reflex from the carotid

sinus a slight swelling at the bifurcation of the common carotid artery at the upper level of the thyroid cartilage The sinus nerve taking its origin in the wall of the carotid sinus is the afferent reflex pathway It joins the glossopharyngeal nerve Irritation of the wall of the carotid sinus causes two reflexes the first produces slowing of the heart while the second causes a fall in the blood pressure The impulses to the heart that bring about the slowing are carried by the vagus nerve

While this patient was in the hospital it was discovered that both attacks of syncope occurred on sultry days when a very closely fitting stiff collar had been worn Inquiry revealed that there had been a considerable gain in weight during the past year and larger collars had not been purchased The patient was accordingly advised to wear a soft type of collar A weight reduction program was advised Large doses of thiamin chloride were prescribed since recent studies have suggested that deficiency states induced by insufficient vitamin $B_1$ are often attended by hyperexcitability of the carotid sinus reflex An abdominal belt was ordered and the patient was given capsules containing 25 mg (3⁄8 grain) of ephedrine sulfate to be taken as needed for vertigo A year later this man reported no recurrence of the syncopal attacks and a weight loss of 10 pounds

Females seem to be less subject to hyperexcitability of the carotid sinus reflex than males[308] Local disease processes most of them arteriosclerotic in nature may produce excessive sensitiveness of this reflex arc by a lowering of the threshold of irritability In some cases where the simpler measures outlined above are not effective in preventing attacks of syncope a denervation of the carotid sinus is to be considered I have seen several cases similar to the one whose history is outlined here but have never been forced to advise surgical relief for the condition

This patient s bradycardia persisted and an average pulse rate of 55 was noted on many occasions subsequently A slow pulse may have no significance particularly in adults who have a hypersthenic build while on the other hand it may serve to attract attention to a serious cardiac defect for example heart block auricular fibrillation or flutter Flutter accompanied by a high degree of block may produce a slow ventricular rate Auricular fibrillation likewise may have a high degree of block which will result in a slow pulse although if carefully studied the pulse will also be found to be irregular

Abnormalities in the formation or conduction of the cardiac impulse may produce a slow pulse Sinus arrest as we have just seen is an example of this kind The pulse may be slow in partial or complete heart block

Certain toxic states may be responsible for bradycardia For example it is often produced by the action of digitalis and it frequently accompanies icterus Following certain infections notably typhoid fever and influenza bradycardia may appear and be quite pronounced The severe toxic state accompanying uremia also produces a slowing of the pulse Intracranial lesions like tumor hemorrhage or concussion commonly cause sinus bradycardia

Bradycardia is frequently met in hypothyroidism or myxedematous states consequently a determination of the basal metabolic rate is indicated in patients who have bradycardia and suggestive clinical findings The low metabolic rate is responsible for the slow pulse rate, both show an increase when thyroid extract is given Finally a slow pulse may be present in patients with aortic stenosis Here a physical examination of the heart serves to differentiate

The management of bradycardia depends upon the cause A slow heart is not necessarily an inefficient heart since many times in cases of complete heart block *the patient can carry on for years with a pulse rate that* is constantly between 30 and 40

### Sinus Arrhythmia

**Case 68** Master R S age six was referred to the Cardiac Clinic of the Woman's College Hospital because of an irregularity of the cardiac rhythm coming on shortly after a tonsillectomy There were no symptoms referable to the heart No murmurs were present and an orthodiagram showed the heart to be well within the limits of normal in size and shape The electrocardiogram (see Fig 194) showed the pulse irregularity to be due to sinus arrhythmia

**Discussion** Sinus arrhythmia following alterations in the vagal tone is the most common irregularity in children Mackenzie referred to it as the youthful type of irregularity Occasionally the condition may be so marked that unless the patient is carefully examined fibrillation may be suspected Sinus arrhythmia may be recognized by noting its relationship to the different phases of respiration It is abolished by atropine which paralyzes the vagal endings in the heart and produces a more rapid regular heart rate Sinus arrhythmia is physiologic, and no treatment of course is required

## PREMATURE CONTRACTIONS (EXTRASYSTOLES)

The occasional occurrence of extrasystoles is compatible with a long and healthy life After 50 they are as insignificant as gray hairs, and if the rest of the examination of the cardiovascular apparatus is negative they should be disregarded Extrasystoles arise from impulses generated outside the sinus node acting on the heart muscle after the refractory period is ended but before the arrival of the next impulse from the pacemaker These beats are easily recognized clinically by noting the occasional intermission in the radial pulse at the same time auscultating over the precordium where the extra beat will be heard

*The premature beat is generally a weaker beat* following the regular contraction It is weak because it occurs at so short an interval after the normal beat allowing the ventricle less than the usual time for filling The output of the heart following the premature beat is less for the same reason in fact some beats may be so weak that the aortic valves do not open The compensatory pause gives ample time for ventricular filling consequently the next beat is stronger than normal and this directs the patient's

attention to the heart and accounts for the complaints of thumping in the chest choking etc

### Origin

Premature beats may originate in any part of the heart the auricular muscle, the ventricular muscle or the A V nodal tissues They may occur at any age from a number of intrinsic and extrinsic causes ranging from a cup of coffee to a coronary occlusion Usually it may be said that the younger the patient the more infrequent is this form of arrhythmia and when it does occur the more likely it is to arise from actual myocardial disease

**Auricular premature contractions** can usually be distinguished from those of ventricular origin by the compensatory pause In ventricular premature beats the pause is fully compensatory if the beat is auricular or nodal the pause is not compensatory

Premature beats may occur infrequently in some patients in which event their recognition is easy In other patients many premature beats may occur in the course of a minute in these cases the irregular rhythm may closely simulate auricular fibrillation Premature beats may occur after each normal beat (pulsus bigeminus) or after each second normal beat (pulsus trigeminus) In rare instances the premature beat may occur between two normal beats without altering the basic rhythm (See Fig 201A) The latter is known as an interpolated beat Here the term, extra systole may be correctly applied

## ILLUSTRATIVE CASES

### Post traumatic Cardiac Neurosis—Significance of Multiple Premature Contractions

**Case 69** Miss E F a typist of 40 complained of frequent pulse skippings following hospitalization for injuries caused by an automobile accident Examination showed a blood pressure of 140/80 frequent premature beats no murmurs and no cardiac enlargement The Wassermann and other laboratory examinations were negative The periods were scant before the accident and following the accident the menses were absent

**Discussion** The discovery of the presence of the premature beats by this patient following an automobile accident was (to use her own words) a terrifying experience When first examined all the chest sensations that usually accompany these interruptions in the cardiac rhythm were described in detail At each subsequent visit the patient brought a detailed record that demonstrated at least an increasing skill in pulse palpation

The age mannerisms menstrual history physical examination type of cardiac irregularity and voluminous charts made of the skippings all suggested the functional nature of the cardiac complaint in this patient Treatment was begun by frankly telling her that no evidence of any

heart disease could be detected outside of the irregularity The premature beats were recorded and the tracing was shown to the patient while the many factors operative in producing them in her case were reviewed She was instructed not to palpate her radial pulse to avoid fatigue, and to get more rest at night To this end she was given a tablet of sodium bromide 1 0 Gm (15 grains) in water after meals Tobacco tea coffee and alcohol were eliminated

The premature beats became much less noticeable and annoying after a week on this regime although examination showed that they were still present At this stage in the treatment the patient was advised to get more out-of-door exercise She chose walks in the country and was not long in noticing that exertion caused the premature beats to disappear The walks were beneficial and she began to sleep better at night and when she was next seen at the end of two weeks improvement was reported A final help was given to her at this time in the form of a capsule of quinidine sulfate 120 mg (2 grains) when it was determined that no reaction followed the initial dose of this drug She was instructed to take one or two of these capsules when the premature beats became annoying during working hour

Search for foci of infection is most important in the management of patients of this type It must not be forgotten that an infected gallbladder can cause these disorders of rhythm which promptly disappear following operation in many instances (Chapter 17)

The relationship between the accident and the onset of the extrasystoles in this patient has medicolegal significance since it brings up the subject of cardiac trauma (page 486) We must admit however that here the stage was set for the occurrence of the premature beats before the accident and trauma should only be viewed as another provoking factor Extrasystoles may follow cardiac injury but they may also occur in the absence of any myocardial damage Their occurrence in this case is by no means proof in itself of damage to the cardiac structure by the accident

### Premature Ventricular Contractions Associated with Hypertensive Heart Disease

**Case 70** Mrs R E a housewife of 60 when first seen complained chiefly of palpitation caused by frequent irregularities in cardiac action The patient was overweight showed an elevation of the blood pressure (190 110) cardiac enlargement of the hypertensive type a systolic apical murmur and beginning congestive cardiac failure The electrocardiogram is shown in Fig 01B

**Discussion** The premature beats also comprised the chief complaint of this patient but examination showed the presence of cardiac damage They were successfully treated by rest and slow digitalization Elixir of phenobarbital (N F ) in 5 cc doses after meals was given for a few days to decrease the apprehension of the patient while measures were instituted for the control of the congestive failure by digitalis With improvement in the circulatory status the premature beats disappeared

# THE PAROXYSMAL TACHYCARDIAS

## A V Nodal Paroxysmal Tachycardia in the Absence of Other Evidence of Heart Disease

Case 71 E W a sales executive of 37 was first seen in November 1934 complaining of attacks of rapid heart action The rate averaged 150 per minute the onset and offset were abrupt and attacks were usually associated with some digestive disturbance Physical examination showed no cardiac enlargement no murmurs and a normal blood pressure The past medical history was negative except for hay fever Wassermann negative The electrocardiogram showed auriculoventricular nodal tachycardia (Fig 206A)

Discussion Paroxysmal tachycardia first described by Cotton in 1867 is caused by a succession of premature beats or extrasystoles emanating from a focus in the auricle the A V node or the ventricle It is a rare arrhythmia occurring only 17 times in 3000 consecutive electrocardiograms at the Woman s College Hospital Of these five were of the auricular variety nine of the nodal and three of the ventricular The auricular and nodal paroxysms occurred in ambulatory clinic patients while the ventricular forms were present in ward patients exhibiting signs of advanced heart disease

In most patients who have the supraventricular types of tachycardia no heart disease can be demonstrated although in some well-compensated rheumatic or arteriosclerotic lesions may be present Occasionally the paroxysms are associated with toxic foci particularly in the gall bladder the elimination of which may be the master stroke in therapy In patients with varying degree of A V block a prefibrillary type of ventricular tachycardia may be observed during Adams Stokes seizures (page 618) Ventricular tachycardia may occur as a sign of the toxic action of digitalis

This patient gave a history of attacks of rapid heart action for a period of years but when their frequency increased he became alarmed and came to the hospital for study The fact that he had no other evidence of cardiac disease was first established The roentgen findings were negative and the electrocardiogram following a seizure (See Fig 206B) was likewise within the limits of normal During his short stay in the hospital many attacks were observed Some were readily controlled by vagus or carotid sinus pressure others showed a tendency to resist all treatment for hours

A striking fact in the past history of this patient was the presence of seasonal hay fever beginning about August fifteenth of each year Paroxysms were more frequent at this time but also occurred at other seasons of the year A history of allergy in these cases warrants a complete study (Chapter 16)

Quinidine One of the most reliable remedies when the paroxysms tend to be prolonged in the absence of organic heart disease is quinidine sulfate the dextrorotary isomer of quinine Wenckebach in 1917 first noted that quinine in malarial patients often abolished an existing fibrillation of the auricles while in 1918 Frey found that quinidine possessed the same gen

eral actions as quinine but exhibited a more intense and selective action on the heart

Quinidine sulfate is a very soluble drug and is rapidly absorbed from the gastro-intestinal tract its action becoming manifest about 20 minutes after administration It is likewise rapidly eliminated a fact which has a direct bearing on the amount and frequency of the prescribed dose

Quinidine lengthens the refractory period of the cardiac muscle and depresses the conduction rate Consequently the drug does not cure disease—it merely smooths out the cardiac action and enables the normal *pacemaker in the sinus node to gain control of the rhythm Quinidine is* therefore useful in paroxysmal tachycardias extrasystoles auricular flutter and in certain selected cases of auricular fibrillation It is most satisfactory in its action when the heart is otherwise sound or the damage if present, is not extreme

Quinidine is best administered in capsule form It is most important to give a test dose of 0 2 Gm (3 grains) of the drug a few hours before therapy is begun unless the emergency is extreme This initial or test dose *in some cases may prove sufficient to abolish the paroxysm* If not in four hours a 0 3 Gm (5 grains) capsule may be given and continued at this interval during the next 24 hours If toxic signs do not appear and the paroxysm persists the dose may be doubled and given at the same interval the next day

Gastro-intestinal tract symptoms may be the first manifestations of untoward effects of quinidine nausea vomiting or diarrhea appear early in some cases Fulness in the head followed by headache, vertigo ringing in the ears and in rare cases deafness are symptoms occasionally seen in some patients in such circumstances the drug should be withdrawn Toxic cardiac symptoms consisting of short runs of tachycardia frequent premature beats or intraventricular block are rare Respiratory distress has been observed to follow large doses Cutaneous eruptions urticarial petechial or scarlatinal in nature have been reported Embolism has followed the ill advised use of quinidine in cases of advanced mitral stenosis in the presence of some degree of congestive failure This complication of quinidine therapy is discussed in connection with the treatment of auricular fibrillation (page 394)

Occasionally quinidine sulfate may be given intravenously if the emergency is extreme in doses of 0 2 to 0 4 Gm dissolved in 30 to 60 cc of physiological saline Some brilliant results have been reported following its administration by this route, but I have never had to resort to it It is almost always possible to give quinidine by mouth in the treatment of paroxysmal tachycardias of auricular or nodal origin

Quinidine sulfate 0 3 Gm (5 grains) every four hours was sufficient to curtail temporarily the paroxysm in this patient We were very much interested in him, and when he left the hospital a few days later he was requested to keep in touch with us and continue the quinidine as a prophylactic measure until he could arrange the time for a more extended

investigation Consequently on his journeys he kept us well informed Communications came from various sections of the country and reflected the attempts made to meet the question of therapy in paroxysmal tachycardia They were written by a patient of high intelligence and I believe the details reported in them to be accurate Therefore I am including the following excerpts unchanged

Boston Mass
November 7 1934

When I left the College Hospital I took a train for Boston Toward morning I had a very severe attack and could not continue at work that day so I went to Dr —— who put electrodes on me again (I am getting used to them by this time) and then gave me two teaspoonsful of wine of ipecac This stopped the attack all right but I vomited for 24 hours I do not feel as if I have any stomach left Perhaps the purpose of this treatment was to get rid of my stomach and drop my heart down in its place in the hope that it would behave better amid unaccustomed surroundings Seriously I would rather have the tachycardia as you call it than vomit my —— head off

The production of vomiting is a most effective way to end an attack Putting the finger down the throat to induce gagging often suffices Apomorphine has been suggested for the same reason In this instance the ipecac was effective when carotid sinus pressure failed The patient s opinion expressed so frankly in the above letter must always be respected before repeating this remedy in the same dosage

Buffalo N Y
November 29 1934

I was fairly well after my vomiting stopped and was able to get some work done I took one of your capsules (quinidine sulfate) after each meal In a week my ears began to ring and when I could stand it no longer I went to see an ear specialist in this city who told me the nerve of hearing had been permanently damaged by the medicine I was taking and advised me to stop it However the ringing continued (It s Armistice Day every day with me) and two days ago I had another attack

The onset of cinchonism was a real indication for the withdrawal of the quinidine Tinnitus and sometimes deafness following quinidine therapy are not uncommon and may appear as in this case at a later date following prolonged administration A drug idiosyncrasy might be expected in an individual of this type although permanent harm rarely results from the use of quinidine Relief has followed cessation of the medication in patients that I have seen Certainly continuous quinidine therapy is not to be recommended for those patients whose attacks occur at long intervals In cases like the one under discussion however I think that it was indicated

because of the frequency of the seizures and the amount of disability they caused

> Chicago Ill
> December 1 1934
>
> The radio business is improving but last week after working for two days straight I had another bad attack When the eye pressure and the neck hocus pocus failed again I did not have the nerve to start up the vomiting so went to see an M D in this city I told him that I had to have relief from the spell and he said that he would use a new drug that would stop it right away It did but let me say that it was some experience! I was given an injection in the arm and in about two minutes I was as red as a lobster and could hardly get my breath I thought that my last hour had arrived and although there is nothing at all unusual about dying in Chicago it seemed a horrible way to meet my end

The new drug mentioned by our trusting traveler was most certainly acetyl beta methylcholin chloride (mecholyl) Popularized by Starr and his workers it has been used in the treatment of the paroxysmal tachycardias since 1933 It is effective on subcutaneous injection in doses ranging from 20 mg (1/3 grain) to 40 mg (2/3 grain) depending on the age and weight of the patient Under 20 years of age an initial dose of 10 mg (1/6 grain) should be sufficient to terminate the attack Obese patients usually require the maximum dosage If the attack continues for a minute after the appearance of the flush described in the above letter (so characteristic of the drug) the site of the injection should be massaged to hasten absorption If this is ineffective the stimulation of the carotid sinus should be tried again for this procedure is sometimes successful if repeated after injection of mecholyl The drug may be given orally in 60 mg to 1 0 Gm (1 to 15 grains) doses which are followed by a slower and milder action and the flushing may fail to appear

Mecholyl occurs as fine white crystals which are hygroscopic and freely soluble in water The solution is stable to heat and has a bitter taste Mecholyl was discovered when search was made for a choline derivative more suitable for clinical use than acetylcholine and lacking its nicotine like action Choline and acetylcholine have been known for many years and recently it has been shown that acetylcholine is the actual chemical substance released in the muscles when parasympathetic nerves are stimulated

Mecholyl lowers the blood pressure slows the cardiac rate causes constriction of the bronchioles (accounting for the asthma) stimulates the sweat glands increases peristalsis, and dilates the peripheral blood vessels Its action in stimulating the parasympathetic nerves may be described a antagonistic to adrenalin The possibility of making to order other similar drugs possessing a variety of such selective actions upon the various nerves

and ganglia of the human body suggests a busy future for the therapeutic engineers

Because of the untoward effects that are sometimes produced by mecholyl a hypodermic syringe containing atropine sulfate 1 mg (1/60 grain) should be kept handy whenever the drug is used Atropine abolishes the effect of mecholyl and in emergencies may be used intravenously The side effects of mecholyl such as salivation sweating and general discomfort Starr considers to be more than compensated for by the relief from the attack I consider them so disturbing that I reserve mecholyl until all other measures to terminate an attack have failed When the prolonged rapid heart rates seriously affect myocardial function I have used mecholyl Each time I have been forced to follow with atropine (page 457)

Mecholyl may cause the inversion of the T wave of the electrocardiogram in some patients When successfully used for an attack of paroxysmal tachycardia the transition from the abnormal rhythm to sinus rhythm is seen This transition may be attended by prolonged conduction short periods of asystole and premature contractions The same effects can be produced by vagal stimulation

Bloom and Cashon[33] observed a very severe reaction following the intravenous injection of 10 mg (1/6 grain) of mecholyl used to stop a paroxysm of auricular flutter Normal rhythm was restored following the appearance of heart block and ventricular tachycardia but they nearly lost their patient Mecholyl should never be used intravenously In cases of ordinary paroxysms that give a previous history of spontaneous cessation where the patient is young and the myocardium in good condition mecholyl should not be used

Let us return from this digression to the recorded experiences of this interesting patient

Los Angeles Cal
February 5 1935

I am sorry not to have written to you before this but have been busy here—in the rain Since it received its volley in Chicago my heart behaved pretty well until two weeks ago After several flare ups I went to an M D in this city and now you will have the real diagnosis! I have a form of intoxication arising in my intestinal tract This doctor has actually visualized the inhabitants and my flora are far from correct There are twice as many germs of one kind present The effect of this inequality in the population in this sector is reflected in my cardiac kick ups I am taking irrigations three times a week for the colon and quantities of a special kind of milk that is charged with reinforcements for the germs that are losing the battle for me Soon all will be right again By the time it stops raining I expect to be cured

Our patient at this stage seems to be in an optimistic mood At least the therapy now seems to be directed toward trying to discover the cause of

the attacks of paroxysmal tachycardia instead of seeking new remedies to abolish individual seizures The presence of hay fever and the occurrence of attacks following gastro-intestinal upsets are more than coincidental My impression was that the offending allergin was a food (Chapter 16) *However, in the form of therapy now described by the patient, the old* idea of auto-intoxication seems to be the guiding principle I could never generate much enthusiasm over the various methods of treatment that have risen to popularity since the *Bacillus bulgaricus* took the stage in spite of the fact that many ills are seemingly cured or avoided and longevity stands as a glowing reward at the end of the trail The one type of intestinal flora may be replaced by another acid producing type but it is a far cry from this fact to the proof that products of intestinal putrefaction are absorbed in sufficient quantities to affect the cardiac mechanism with the production of paroxysms of tachycardia

St Louis, Mo
April 15 1935

I am still able to keep going but am definitely finished with all doctors My attacks after all the treatment I have received in different cities of the country are about the same as they were when I stopped in to see you in Philadelphia My heart continues its temperamental career It does its stunts at the most inconvenient times Last week I had a spell during an examination for life insurance and the company would not issue a $10 000 increase I have spent a sizable sum on doctors during the past year and if all the electrocardiograms I have had taken were collected and placed end to end they would form a cardiac race course reaching from here to Philadelphia I am going to be my own doctor for a while for I certainly cannot be much worse than I am now

A discouraged patient! However it is not entirely the fault of the physicians he has seen for he has not stayed in one place long enough for a thorough study to be made The therapy has been mainly for the seizures and the patient s manner of living in itself plays no small part in producing these frequent recurrences

Cincinnati Ohio
June 19 1936

No doubt you have forgotten all about me by this time but I think that you ought to know how I finally obtained relief from my attacks I did not take any medicine or see any doctors for six months after I last wrote to you I had to continue at work but the attacks came on so often that I was quite miserable most of the time and lost considerable weight Last October while I was motoring through a small town in Ohio I decided to apply for medical aid once more This time instead of choosing a specialist who would have taken another dozen feet of film of my

heart beat and have gazed at me through a fluoroscope I walked into the office of a general practitioner I told my story again and the old man was able to see me in one of my spells He then reached in the drawer of his desk and from among other things pulled out a handful of digitalis pills (that you had always told me I did not need) and advised me to take one after each meal for five days and then one every day He told me to take a two weeks vacation and return at the end of that time to see him I followed his instructions to the letter chiefly because he did not order a couple of dozen laboratory tests and puncture me with a hypodermic needle or try to force an entrance into some region of my body with a pipe line During those two weeks I did not have a single attack When I saw him again he told me to take one of the pills every day and go back to work I have not had a spell for eight months and I am sure that I am cured

Many times I have advised my surgical friends not to administer digitalis when their patients emerging from the anesthetic happen to have one of these attacks of paroxysmal tachycardia to which they may have been subject for years I try to demonstrate the quiet breathing the lack of any sign of congestive failure the dry lung and the normal temperature Even the serene countenance of the patient fails to convince the worried surgeon and when I return to the ward the next day I generally note that digitalis has been continued until the abrupt cessation of the abnormal rhythm Post hoc ergo propter hoc is a belief not entirely confined to lay circles However I still preach the doctrine that digitalis is poor therapy for this group of patients who have paroxysms of tachycardia when they have otherwise normal hearts A letter like this one forces me to admit the exception In a small group when all other measures fail complete digitalization with the continuance of maintenance dosage may bring relief The country doctor tried this and succeeded He was most up to-date in his method of using digitalis and in establishing proper maintenance allowance over a long period He was also able during the course of one visit to gain the patient s confidence and insist on a vacation The rest played no small part in the cure

Paroxysmal tachycardia is rarely met in children Tarran and Jennings[37] report 52 cases in literature from 1892 to 1935 in patients under 15 In a patient recently reported by Wright[4 3] 17 paroxysms were observed in a child of six years who had an otherwise normal heart Many of these were immediately terminated by 5 mg doses of mecholyl administered hypodermically There was a strong emotional factor in this Jewish child inasmuch as the paroxysms did not occur while she was in a convalescent hospital but immediately reappeared upon her return to the environment of her home I recently observed a paroxysm of auricular tachycardia in a Jewish child of five that lasted two weeks and resisted all forms of therapy that have been described Studies made during and after the

seizure showed a normal heart Food allergy was the background suspected in this case

### Paroxysmal Ventricular Tachycardia Complicating Acute Coronary Occlusion

**Case 72** F C an unemployed accountant of 56 was well except for the presence of elevated blood pressure for some years until an hour before admission to the hospital At this time following a heavy meal he had a sudden attack of severe precordial pain radiating to both sides of the neck It was promptly followed by vomiting and collapse When seen in the receiving ward the patient was in shock with a pulse so rapid that it was impossible to count the rate accurately Death occurred while the tracing shown in Fig 245 was being taken

Clinical Diagnosis A Etiologic Hypertension B Anatomic Cardiac enlargement Acute coronary occlusion C Physiologic Paroxysmal ventricular tachycardia D Functional Classification Class 4

Discussion Paroxysmal ventricular tachycardia is a much more serious arrhythmia and generally occurs in the presence of grave heart disease It is not infrequently associated with coronary accidents similar to the one described above Slight irregularities in the rhythm may serve to make the diagnosis at the bedside and differentiate from paroxysmal auricular tachycardia Paroxysmal auricular tachycardia is characterized by its absolute regularity Careful auscultation in patients with ventricular tachycardia may also reveal alterations in the heart sounds that are important in the differential diagnosis In ventricular tachycardia the first heart sound will occasionally vary in intensity It may be louder with some beats and reduplicated in others due to the varying positions of auricular systole in the cardiac cycle

Vagal stimulation and the other measures suggested for paroxysmal auricular tachycardia have no effect in ventricular tachycardia Digitalis is valueless in fact it may even increase the rate in some cases The drug of choice in this emergency is quinidine It is effectual because the arrhythmia is in all likelihood caused by a circus movement in the ventricle This mechanism may be rapidly fatal unless treatment is prompt In these emergencies quinidine should be given intravenously (page 384) before the sudden burden of the arrhythmia on an already damaged myocardium terminates the picture

## AURICULAR FLUTTER

In auricular flutter the impulse for cardiac contraction arises from a wave that follows a regular path around the auricular musculature at a speed of 260 to 310 revolutions a minute The pulse rate in flutter depends upon the ability of the ventricles to respond to the rapid succession of stimuli it receives from this abnormal auricular wave At times this response is one to one but more commonly a ventricular contraction follows the stimulus of every second or third revolution of the circus movements in the auricle The onset of flutter is abrupt It may persist for a short while as a paroxysm or may continue unchecked for months or years

When flutter disappears it may be replaced by either fibrillation or normal rhythm

Flutter is a rare disorder encountered only 20 times in the first 3000 electrocardiograms taken at the Cardiac Clinic of the Woman s College Hospital Flutter may be observed in otherwise normal hearts or may complicate rheumatic arteriosclerotic or thyroid heart disease Occasionally other toxic states of the myocardium may be responsible for its appearance

The pathologist finds no characteristic auricular lesion in cases of flutter but this should not be surprising inasmuch as it is usually brought about by a reduction in the refractory period of the muscle which permits continuous passage of the original contraction wave The clinical symptoms produced by the sudden onset of auricular flutter vary Rarely there may be none at all and the condition may be brought to light at a routine examination In other cases the ventricular rate is too rapid to count particularly where a one to one response occurs In these cases the tax on the cardiac reserve is great and if myocardial disease is present signs of congestive failure may appear rapidly Unconsciousness from cerebral anemia in some cases has been reported Fortunately the ventricles do not respond to each stimulus but to every second or third revolution of the flutter wave in the auricle in which event the pulse rate will be about 150 beats per minute The response of the ventricle will occasionally vary every second third or fourth stimulus getting through the junctional tissues and producing an irregular pulse resembling fibrillation Usually however the pulse is rapid and regular When the attack is over the ventricular rate will fall suddenly to normal and any symptoms that may have been present will disappear quickly

A history of a rapid ventricular rate in a middle aged or elderly patient sudden in onset and present for some weeks suggests auricular flutter If carotid sinus pressure slows the ventricular rate the diagnosis can be made with certainty Exercise may speed up the ventricular rate in flutter and make the rhythm regular the opposite is true in fibrillation Flutter can be distinguished from a paroxysm of tachycardia on the basis of the ventricular rate The simple paroxysmal tachycardias usually have higher ventricular rates (180 to 230)

## ILLUSTRATIVE CASES

### Paroxysmal Auricular Flutter in an Otherwise Normal Heart

**Case 73** H T a male laborer of 46 was first seen January 10 1935 complaining of vertigo and precordial oppression accompanying the sudden onset of a rapid heart rate Except for the history of peptic ulcer the patient had been well until a month before the onset of the attack when he developed pains in the shoulders arms and back. Although vigorously treated by his physician with salicylates and baking he became worse and was unable to work On purely empirical grounds he was placed on sulfanilamide therapy After three doses of 1 Gm each he developed nausea slight cyanosis and sudden rapid cardiac rate On admission the pulse was 150 in all positions More rapid auricular pulsations were noted in the neck veins The heart size was not increased and the sounds

were of good quality No murmurs were heard The blood pressure was 100/80 Wassermann blood sugar complete blood count and blood urea were normal

CLINICAL DIAGNOSIS A Etiologic Unknown (Toxic from sulfanilamide?) B Anatomic No cardiac enlargement C Physiologic Auricular flutter D Functional Classification Class I Therapeutic Classification Class C

**Discussion** On admission the tracing (Fig 143B) showed typical auricular flutter with a two to one ventricular response The patient was in

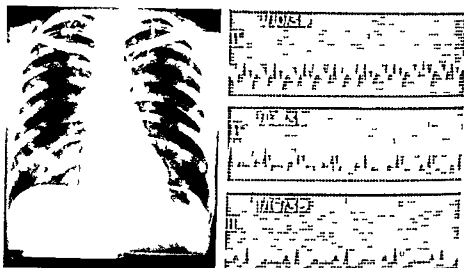



FIG 143 A Roentgenogram of chest The cardiac silhouette is normal in size and shape B The electrocardiogram Examination made on 10/1/39 shows presence of auricular flutter with a 2:1 ventricular response The next strip from the same lead three days later following the administration of 0 9 gram (13½ grains) whole leaf of digitalis Three days later (1/16/39) after 1 8 grams ( 7 grains) of digitalis whole leaf had been given the drug was discontinued Normal rhythm is now present Note depression of the S T intervals

good condition in spite of the continuation of the rapid ventricular rate, consequently it was decided to digitalize him slowly The next tracing was taken three days later after nine tablets each containing 100 mg (1½ grains) of whole leaf of digitalis had been given When a total of 18 tablets had been given the digitalis was stopped, and normal rhythm appeared spontaneously

Administration of digitalis usually abolishes auricular flutter by converting it into auricular fibrillation In this patient we see a return to normal rhythm without the intervening fibrillation However there may have been a very short period of fibrillation following the disappearance of the flutter that was not observed Since large doses of digitalis are needed to produce an effect in the presence of auricular flutter, the patient should be kept under constant observation while the drug is being given Warnings such as anorexia and a slight nausea may be overlooked, but the ap

pearance of frequent premature beats in the tracing should always be considered a signal of the approach of dangerous toxic rhythms. The digitalis should then be stopped. Normal rhythm usually returns. If there is no restoration of sinus rhythm in one week in patients who show no signs of mitral disease or auricular enlargement, quinidine may be considered. The patient now under discussion would have been an excellent case for quinidine therapy if normal rhythm had not returned following digitalization. Likewise, if forced to abandon the use of digitalis because of the appearance of toxic symptoms, quinidine may be begun in a few days. After the usual preliminary test dose of 0.2 Gm. (3 grains), a 0.3 Gm. (5 grains) capsule is given every four hours (four times daily). If this does not restore normal rhythm in three days, it can be given five times daily. If there is still no effect, the dose can be increased every two days until a total of 2.6 Gm. (40 grains) a day is reached. In extreme emergencies where the abnormal rhythm is threatening life, this amount may be exceeded in an attempt to restore sinus rhythm and save the myocardium. In less urgent cases it is not wise to exceed 2.0 Gm. to 2.6 Gm. (30 to 40 grains) of quinidine daily. Where the paroxysm of flutter is successfully abolished by the use of quinidine, it is essential to continue the drug in maintenance dosage of 0.3 Gm. (5 grains) three times daily. In patients who have mitral disease and a large left auricle, it is a wiser course to accept the fibrillation and efficiently control the ventricular rate by digitalis.

With the exception of a report of a transient nodal rhythm by Dozzi[81] following an initial dose of 5.4 Gm. (80 grains) of sulfanilamide, I am not aware of any reports in the literature of the appearance of toxic rhythms following this commonly used drug. Considering the amount of the drug that must be consumed by all types of ambulatory and hospitalized patients, if cardiac manifestations are common, we would have seen more instances during the past years. This patient and the patient of Case 77 are the only ones that I have encountered where the relationship between the onset of the arrhythmia and the exhibition of sulfanilamide suggested the possibility of toxic action. In both cases the appearance of a paroxysm closely followed ingestion of the drug, and in each instance the paroxysm was the only sign of heart disease encountered after complete studies were made. Certainly fear of any cardiac complication should not deter us in the rational use of sulfanilamide or its derivatives.

### Auricular Flutter Accompanying Rheumatic Heart Disease

**Case 74.** Mrs. E. G., a housewife of 29, when first seen was complaining of palpitation and rapid heart action. She gave a history of two attacks of rheumatic fever. Examination showed a pulse of 150, B.P. 110/74. The orthodiagram suggested the presence of mitral stenosis and regurgitation, although the physical signs were not conclusive in the presence of such a rapid cardiac rate. The electrocardiogram showed auricular flutter (see Fig. 28).

Clinical Diagnosis. A. Etiologic: Rheumatic (Inactive). B. Anatomic: Cardiac hypertrophy; Mitral stenosis; Mitral regurgitation. C. Physiologic: Auricular flutter. Functional Classification: Class 1. Therapeutic Classification: Class C.

**Discussion** When this patient was first examined her ventricular rate was 150 There was no decrease in her exercise tolerance the lung fields were clear and her color was excellent She was given a tablet of the whole leaf of digitalis 0 1 Gm (1½ grains) after each meal for one week (weight 140) and was allowed to be out of bed but not out of the house during this time At the end of one week the pulse was slower and totally irregular An electrocardiogram (see Fig 228B) showed that fibrillation had replaced the flutter consequently the digitalis was entirely withdrawn and the patient instructed to return in another week At the third visit normal rhythm was present (see Fig 228C) At least half of the cases of auricular flutter when similarly treated will show a return to normal rhythm in this classical fashion

If advanced mitral stenosis is present and normal rhythm does not appear it is far better to continue the digitalis in daily maintenance dosage particularly if the ventricular rate is well controlled rather than attempt to restore normal rhythm by the use of quinidine If successful and sinus rhythm returns it soon gives way again to flutter or fibrillation so there is less risk if the ventricular rate of fibrillation is reduced and maintained at 70 beats per minute by digitalis

When this patient was examined a year later flutter had returned (Fig 228D) This time when fibrillation was produced, the digitalis was continued in maintenance dosage The rest of this patient s treatment was governed by subsequent developments in the course of her rheumatic heart disease (Chapter 3)

## AURICULAR FIBRILLATION

The most common cause of a perpetual arrhythmia is auricular fibrillation When viewed in the laboratory animal the fibrillating auricles show no co-ordinated systole twitchings and undulatory movements suggest incessant activity of the entire musculature produced by the irregular course of the fibrillation wave In flutter (see Fig 225) the pathway of the circus movement is the same at each revolution In fibrillation (see Fig 229) the course varies and impulses arrive at the A-V node at irregular intervals This accounts for the total irregularity of the rhythm in fibrillation

Auricular fibrillation in young people generally complicates the later course of mitral stenosis As a rule care should be used in making the diagnosis of fibrillation in patients less than 15 years of age although at times this arrhythmia may complicate congenital lesions (page 550) In later life auricular fibrillation frequently accompanies arteriosclerotic or hypertensive cardiovascular disease Rarely is it associated with aortic regurgitation and for this reason is seldom met in syphilitic heart disease

Recognition of auricular fibrillation usually presents little difficulty If the rhythm is irregular and the rate exceeds 120 per minute this arrhythmia is almost certain to be present If digitalis has slowed the ventricular rate

to 60 or below the diagnosis may be more difficult However it is well to remember that exercise increases the heart rate and accentuates the irregularity when auricular fibrillation is present

## ILLUSTRATIVE CASES

### Rheumatic Heart Disease with Auricular Fibrillation and Congestive Failure

**Case 75** V P a white American janitor of 4 had a single attack of rheumatic fever in 1928 at th age of 30 He was well until the summer of 1938 when he noticed increasing dyspnea on stairs This became worse until three months later edema of the feet appeared at night When first seen in February 1939 there was orthopnea and anasarca and the pulse was totally irregular with a precordial rate of 150 The heart was increased in size in all diameters and there was a systolic and a diastolic murmur at the cardiac apex The pulmonic second sound was accentuated

Clinical Diagnosis A Etiologic Rheumatic Inactive B Anatomic Cardiac hypertrophy Mitral stenosis Mitral insufficiency C Physiologic Auricular fibrillation Congestive cardiac failure D Functional Classification Class 4 Therapeutic Classification Class E

Discussion In this patient the onset of congestive failure was attended by a change from sinus rhythm to auricular fibrillation The ventricular rate was rapid and consequently the stage was set for a good therapeutic result In patients who have cardiac failure and rapid auricular fibrillation a great deal may be expected from the prompt use of digitalis in full dosage The bombardment of the responsive ventricle by the impulses from the fibrillating auricle is lessened many of the inefficient feeble beats are eliminated the pulse deficit disappears diuresis begins and the signs of cardiac failure recede Subjective improvement parallels the changes produced by this drug and the patient is pleased mightily

Examination of this patient five days after the administration of 1 3 Gm (20 grains) of digitalis whole leaf showed continued fibrillation but a much slower ventricular rate No attempt was made to restore normal rhythm because of the extent and type of the lesion When the ventricular rate reached 70 the digitalis was cut to 0 1 Gm (1½ grains) of the whole leaf daily This maintenance dose will be necessary for the rest of the patient s life One injection of mercupurin 2 0 cc was given at the start of the treatment to help the digitalis clear the edema The same result would have been achieved eventually without the mercupurin in this patient

### Paroxysmal Auricular Fibrillation—No Other Evidence of Heart Disease

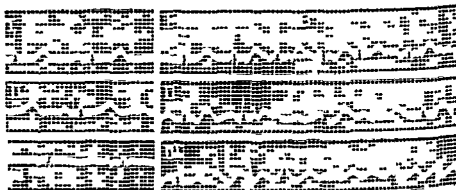
**Case 76** Mrs A C W an American house ife of 61 was first seen n 19 9 with the chief complaint of attacks of rapid irregular heart act on These had been experienced on the a erage of three times a year for the past 14 years

Physical Examination BP 1 8/80 The heart was normal in size and shape (Fig 144A) There were no m rmurs Other features of the exam nat on were irrelevant except for the presence of a small calc fied fibroid tumor At the time of the next seizure an electrocard og am was obtained (Fig 144B) which showed the p esenc of aur cula

fibrillation Normal rhythm returned spontaneously one hour later (Fig 144B) From 1939 until 1940 many attacks similar to this one have been observed There are no signs of *congestive failure during the attacks The heart has not increased in size* as shown by frequent orthodiagraphic studies The basal metabolic rate has been taken many times, the figures ranging from minus eight to plus four



A



B

Fig 144 A Roentgenogram The heart is not enlarged B The electrocardiogram (a) normal rhythm (b) taken during a paroxysm Auricular fibrillation is present

Discussion The occurrence of paroxysmal auricular fibrillation in *patients exhibiting no other signs of cardiac disease is relatively rare* In a study of 200 cases of paroxysmal auricular fibrillation Parkinson and Campbell[293] found that 9 per cent showed no apparent cause for the occurrence of this marked arrhythmia White and Jones[393] in an analysis

of 376 cases of auricular fibrillation found that 80 per cent were free from heart disease

In the five patients that I have studied the inciting factors in precipitating the paroxysm of fibrillation were alcohol in two a gastro-intestinal upset in two and unusual exertion in one In the patient I have selected for discussion here the attacks have been present for 25 years and always follow a digestive upset

Since there are no gross or microscopic lesions of the auricular musculature characteristic of fibrillation we may assume that the presence of the arrhythmia does not depend upon a definite structural defect in the heart It is primarily a functional alteration and may be produced in many instances by extracardiac neurogenic forces

Hyperthyroidism is the first thought when no explanation for the paroxysms of fibrillation is evident at the time of the initial study Many cases will ultimately fall into this group particularly if the fact is kept in mind that thyrotoxic heart disease may exist in the presence of a normal basal metabolic rate A complete study should be made of the skin the eyes the neck the weight changes and the hands for the presence of tremor before deciding against the thyroid as the dominating influence The physical signs should be weighed with the laboratory evidence

When paroxysmal auricular fibrillation is present the heart should be carefully watched between attacks Evidence of mitral stenosis should be sought in the character of the first heart sound and the presence of early diastolic murmurs and the size and shape of the heart on fluoroscopic examination A large auricle in one of the oblique positions on fluoroscopy may point to an overlooked mitral lesion

All of the patients that I have studied who have suffered from paroxysms of auricular fibrillation with no other evidence of heart disease have had one thing in common This was a marked nervous instability All were emotional and showed a tendency to over reaction to the ordinary stimuli of life In each instance I suspected that the trigger mechanism was the thyroid gland but could not prove this view to be correct It is certainly true that this type of person presents much more opportunity for the occurrence of abnormal stimuli that are reflected in the cardiac mechanism by the sudden onset of paroxysms of auricular fibrillation How the irregular circus movement in the auricle is started in these cases remains unknown It must be closely related to the mechanism operative in disturbances of the thyroid gland The trigger may be the adrenal

In the patient under discussion the investigation was first directed toward the thyroid status Since repeated determinations of the basal metabolic rate showed no elevation and a therapeutic trial of iodides was entirely ineffectual in lessening the frequency of the seizures studies were carried further and an allergic cause was sought

While I have seen allergy assume an important etiologic role in a few cases of paroxysmal auricular tachycardia I have never met it as the exciting factor in paroxysmal auricular fibrillation However this patient

was completely studied by Dr Kern The result of his investigation and a discussion of the case from the allergic standpoint will be found on page 459

Quinidine in 0 3 Gm (5 grains) doses was found to be invariably successful in restoring normal rhythm in this patient Consequently when attacks appeared with increasing frequency she was placed on maintenance dosage of the drug as a prophylactic measure A 0 3 Gm (5 grains) capsule was given twice daily and later this was increased to three times daily While occasional small doses of quinidine were very well tolerated when maintenance doses were begun minor symptoms were complained of at the end of the second week These consisted of headache vertigo nausea and finally tinnitus When the maintenance dosage was decreased paroxysms returned and larger doses were found to be required to restore normal rhythm The patient finally requested that she be allowed to take the quinidine at the time of the paroxysms and stop the daily ration of the drug She stated that she would rather run the risk of an attack at an inconvenient time than tolerate the annoying symptoms that accompanied maintenance doses of quinidine

### Congestive Cardiac Failure Induced by a Paroxysm of Auricular Fibrillation in a Senile Heart

**Case 77** W M a male clerk of 70 when first seen was complaining of dysuria frequency and nocturia of three days duration The temperature was 101 F pulse 90 and regular and the heart was normal in size and shape There were no murmurs The urine showed a light cloud of albumin and was loaded with pus and blood cell When an unidentified streptococcus was cultured from the urine a diagnosis of urinary tract infection was made and the patient was given 0 Gm (30 grains) of sulfanilamide as an initial dose and 1 0 Gm (15 grains) every four hours When examined the next day the pulse was 140 and totally irregular Orthopnea and cyanosis were present Rales were heard at the lung bases

**Discussion** Paroxysms of auricular fibrillation not uncommonly follow toxic states for example those produced by infectious diseases like pneumonia or the ingestion of a drug or poison in large quantities In this old gentleman it was difficult to decide whether to associate the onset of auricular fibrillation with the toxemia of the urinary tract infection or the sulfanilamide

The therapy employed was based on the signs of congestive failure that appeared so quickly dyspnea cyanosis and finally pulmonary edema The patient had not previously been taking digitalis consequently an initial dose of strophanthin 0 4 mg (1/150 grain) was given intravenously and in addition a hypodermic injection of 15 mg (¼ grain) of morphine sulfate There was marked improvement in the congestive signs in two hours At this time tablets of digitalis whole leaf were started by mouth In 24 hours normal rhythm returned

In Case 76 the paroxysms of auricular fibrillation caused complaints of palpitation and vertigo but no signs have ever appeared that point to the slightest circulatory insufficiency Consequently when we see how well

the heart muscle has withstood these repeated assaults for many years it is small wonder that we speak well of its functional capacity and doubt the presence of cardiac disease

In Case 77 however the situation is different In this old man the sudden burden of the arrhythmia overtaxed the myocardial capacity and precipitated signs of failure The intravenous administration of strophanthin was called for in such an emergency and it was followed by rapid improvement Digitalization was completed using the whole leaf tablet by mouth and maintenance dosage was then continued

## HEART BLOCK

This arrhythmia was recognized and described clinically before the specialized tissue of the conduction system was discovered The work of many investigators during the last part of the nineteenth century proved that impulses are conducted from the auricle to the ventricle over the A V or His bundle If the tissue of the normal bundle is clamped lightly in the experimental animal the function is interfered with and the length of time for an impulse to pass from auricle to ventricle is increased If the clamp is tightened the time is still further lengthened until an occasional beat is dropped Further compression will cause higher degrees of block until the process is complete When this occurs the auricles continue to beat at their usual rate while after a pause the ventricles respond to a new (or idioventricular) center just below the level of the lesion and resume their contractions at a much slower rate During the time of ventricular adjustment cerebral anemia usually occurs If the pause is longer it may be attended by coma and convulsions A similar sequence of events has been observed clinically when conduction through the bundle of His has been seriously affected by a disease process The attacks of cerebral anemia may cause vertigo and faintness or may be attended by coma and convulsions These attacks have been termed Adams Stokes seizures although Adams and Stokes were by no means the first to describe them

Since the electrocardiograph records faithfully the time consumed by the passage of the contraction impulse from auricle to ventricle slight delays are readily detected by this method (page 615) The presence of even a mild degree of inflammatory change in the neighborhood of the bundle or interference with a portion of the coronary blood supply may be reflected at once in the prolongation of the P R interval of the electrocardiogram Many times this is the *only sign present to suggest cardiac* involvement during the course of acute rheumatism or diphtheria The prolonged P R intervals may be missed since few patients are studied by the electrocardiographic method However when the heart block progresses to the stage of dropped beats or to complete dissociation it will attract clinical attention if the pulse is carefully studied

The anatomic change present in infectious states may be a temporary one and complete functional recovery of the conduction system may be

possible following resolution of the inflammatory exudate The electrocardiogram will then return to normal Temporary change in the conduction time may also be associated with excess vagal stimulation, asphyxia uremia or it may follow the toxic action of certain drugs like digitalis or quinidine Coronary disease particularly occlusion may cause a more permanent alteration to occur although even here collateral circulation may *make possible the return of normal function in a few days (see Fig 243)* Infiltration of the conduction tissues by a secondary tumor growth may produce heart block of a more permanent nature because of destruction of the tissue (page 407) Heart block rarely results from congenital deformities in the septum (page 543)

The gumma of syphilis as a cause of complete heart block is most infrequent although this is generally the first one mentioned by the student Invasion of the bundle from the vegetative growths of subacute bacterial endocarditis is likewise unusual Graybiel and White[141] in a recent survey of 72 cases of complete A V dissociation found the cause *to be coronary disease in 47 congenital heart disease in 4 possible con*genital heart disease in 2 rheumatic heart disease in 3 syphilis in 3 diphtheria in 4 while the remaining 9 cases were of mixed or uncertain etiology

At postmortem when sections of the heart are made and the conduction system is inspected the degree of change by no means parallels the clinical symptoms For example only a thin strand of tissue may be found remaining yet during life the conduction system may have shown perfect function In other cases a great deal of apparently good tissue may be seen in hearts of patients who showed complete block during life The *only explanation in the latter instance seems to be the chemical changes* present during life in and about the bundle tissue caused by anoxemia and circulatory stasis In cases where no change at all is seen in the bundle tissue and complete block was known to have been present we must consider vagal action and the effect of certain drugs

While the electrocardiogram may show a prolongation of the P R interval as the only evidence of an acute myocarditis two physical signs that will aid in establishing the diagnosis may be detected by the careful clinician If the time interval between the auricular and ventricular systole is lengthened a splitting of the first heart sound may occur The first element of this sound is caused by auricular systole Again if a mitral stenosis is present the characteristic rumbling murmur may change its position from presystole to early or mid-diastole for the same reason Where the degree of block is greater and occasional dropped beats occur the pauses that accompany them may be detected in the radial pulse and the differential diagnosis from premature beats may be made by careful auscultation Close inspection may reveal a wave in the jugular veins produced by the blocked auricular contraction during the period of ventricular asystole

Auriculoventricular block is a symptom and not a disease Consequently in most of the following illustrative cases it will be noted that the treat

ment is that of the underlying condition causing the block However when the block is complete and causes a profound disturbance in the cardiac action with Adams Stokes seizures it requires special treatment In White s series of 72 cases symptoms related to the block itself occurred in 44 and in four instances were the probable cause of death

## ILLUSTRATIVE CASES

### Prolongation of the P R Interval (First Stage Heart Block) During the Course of Acute Rheumatic Infection

**Case 78** C C a male elevator operator of 23 was admitted to the Woman s College Hospital on May 7 1936 complaining of backache and pains in the joints The onset of the present illness was a week prior to admission when a sore throat and evening temperature appeared Physical examination on admission showed T 100 B P 116/76 pulse 88 rhythm regular no cardiac enlargement and no murmurs The joints showed no swelling or redness An electrocardiogram showed prolongation of the P R intervals to 0 3 second

Clinical Diagnosis A Etiologic Rheumatic Active B Anatomic No cardiac enlargement C Physiologic First stage heart block D Functional Classification Class 1 Therapeutic Classification Class E

**Discussion** The prolongation of the P R interval in this patient was a very valuable finding suggesting at once the nature of the joint manifestations An involvement of the heart by the rheumatic process was suspected and a diagnosis of acute myocarditis based on this finding The treatment of the heart block in this instance is that of the underlying rheumatic process (Chapter 3)

### Complete Heart Block Complicated by Adams Stokes Seizures

**Case 79** Mr O F an executive of 74 was first seen October 10 1936 complaining of weakness dyspnea and vertigo Hypertension had been present for some years Three months previous to the first examination marked edema of the feet appeared but cleared up on bed rest The dyspnea persisted to the time of the first examination

Physical Examination The blood pressure was found to be 180/110 Pulse 40 The heart was enlarged to the left on percussion the left border measuring 1 5 cm from the mid sternal line and the right border 3 0 cm An apical systolic bruit was heard poorly transmitted in the direction of the axilla The liver edge was palpable beneath the right costal margin There was no edema

Laboratory Data The first electrocardiogram (see Fig 213) taken 10/10/36 showed complete dissociation with auricular rate of 100 and ventricular rate of 40 Occasional ventricular complexes showed the presence of faulty intraventricular conduction

An orthodiagram showed a heart of hypertensive shape with a cardiothoracic ratio of 0 51 It was possible to note the A V dissociation during fluoroscopic study

Course The patient was examined again four months later (February 1937) at which time he still complained of weakness and dyspnea In addition he stated that he had experienced two spells of sudden syncope both following emotional upsets Consciousness was lost on each occasion for less than one minute There were no convulsive movements and recovery was prompt and complete

An electrocardiogram taken on the second visit showed striking differences in the T waves in both direct and indirect leads There had been no precordial pain or paroxysmal dyspnea nevertheless these changes were ascribed to a coronary occlusion which had occurred since the last examination The orthodiagram showed no essential

change The urine showed a trace of albumin fixation of the specific gravity (1 008) and both hyaline and light granular casts The blood Wassermann reaction was negative and the blood pressure reading was increased this time measuring 220/110

The next examination was made 15 months later at the patient's home (May 20 1938) His interval history indicated a steady downhill course During the late winter and early spring months his spells of syncope had increased in frequency They were of longer duration and convulsive twitchings were reported to have taken place during all the seizures There was no incontinence of urine or feces Members of the family who witnessed attacks stated that they were preceded by pallor However the onset of each attack was usually announced by the patient himself Cyanosis followed the pallor and the respirations became frequent deep and noisy In a minute convulsive twitchings of the extremities appeared followed by return of consciousness We were able during the next 24 hours to witness many of these seizures They varied in length from less than a minute to over six minutes During the early shorter seizures the patient would recover and resume the conversation Electrocardiograms were continuously recorded The next day the attacks were longer in duration and consciousness returned more slowly Finally after an attack lasting six minutes consciousness was not regained During a terminal coma brief seizures continued until death nine and one half hours later Postmortem examination was not obtainable

CLINICAL DIAGNOSIS A Etiologic Hypertension Arteriosclerosis B Anatomic Cardiac enlargement Coronary occlusion Relative mitral insufficiency C Physiologic Heart block with Adams Stokes seizures D Functional Classification Class 3 Therapeutic Classification Class E

**Discussion** The Adams Stokes attacks in this patient from the time of their onset 15 months before death appeared to have a direct relationship to emotional upsets During the last 48 hours of life these attacks varied in duration from one-half minute to six minutes becoming more prolonged and more frequent after the first 24 hours until the seizure that lasted over six minutes permanently eliminated consciousness Until the last 24 hours of life the patient was able to announce the beginning of every attack which always coincided with the disappearance of the radial pulse Pallor followed and the respirations became deep and noisy There were no convulsive movements noted at this time although the muscles about the neck and face seemed to be tighter The pallor continued changing in less than a minute to cyanosis When the pulse beats were felt the patient's face became purplish red and convulsive movements occurred Apnea followed the color change in the face

The series of events observed in this patient during successive Adams Stokes attacks is not fortuitous but dependent on a definite relationship between respiration and circulation that Formijne[103] has observed in a series of clinical and experimental studies With the onset of the attack all blood flow ceases Respirations continue giving the blood stagnant in the lungs a chance to become more completely saturated with oxygen and to lose a great deal more of its carbon dioxide than ordinarily occurs With recovery the first substantial cardiac contraction sends out this pooled hyperventilated blood Apnea results when it strikes the respiratory center The patient's color undergoes a marked change with the arrival of blood rich in oxygen in the capillaries of the face The convulsive movements that follow at this point arise from the alkaline state of the blood

since the hyperventilation received in the lungs during the cessation of circulations washed out an excess of carbon dioxide

A study of this patient s electrocardiograms before the seizures suggested a coronary accident as the cause of the complete block The attacks were preceded by the appearance in the tracing of occasional ventricular extra systoles which became more frequent and then appeared in short runs of three or more *leading into the attack A very rapid ventricular rhythm* was seen during some of the periods of unconsciousness During others stand still of the ventricles was observed

### Treatment

The proper management of these attacks depends upon the electrocardio graphic findings Where ventricular standstill causes the cerebral anemia and syncope the treatment consists of the intramuscular injection of ½ to 1 cc of a solution of epinephrine hydrochloride U S P between attacks to stimulate the idioventricular center While this drug is useful in pre venting seizures it should be administered with great care Some observ ers[114] have successfully combined epinephrine with barium chloride The latter drug increases the irritability of the area in the ventricle below the level of the block and makes it more susceptible to the action of the epinephrine In the presence of complete standstill of the ventricles the epinephrine should be given by intracardiac injection[30, 334] inasmuch as action of the drug by any other route is unlikely during the period of cardiac standstill Epinephrine tends to increase both auricular and ven tricular rates in these instances

While a life saving procedure in the presence of standstill epinephrine may be actually harmful if the prefibrillary type of ventricular tachycardia or ventricular fibrillation are present (see Fig 214) These abnormal rhythms are evidence enough of the irritability of the tissues An accentua tion of this state by the introduction of epinephrine is contraindicated since it has been shown[1 3 335] to produce or prolong ventricular fibrilla tion which is likely to result in death

The use of ephedrine in complete heart block was first reported by Miller [71] His patient had complete A V dissociation but no Adams Stokes seizures and showed increase in auricular and ventricular rate and a change in the shape of the P waves and ventricular complexes following hypo dermic injection of 100 mg (1½ grains) Stecher[3 3] reported favorably on ephedrine when used in a similar case having Adams Stokes seizures fol lowing ventricular standstill The drug was given by mouth in 30 mg (½ grain) doses three times daily for one week and then 20 mg (1/3 grain) three times daily for two weeks During this time there was complete relief from attacks Cheer Tung and Bien[55] found that patients with complete heart block responded to ephedrine quite differently and suggested the administration of atropine to prevent the reflex stimulation of the vagus and the rise in blood pressure following ephedrine The combination of these two drugs does not abolish the complete block

Wedd recently reported a case of complete heart block resulting from rheumatic infection in a woman 41 years old Several Adams Stokes seizures were observed and epinephrine was without effect Following the injection of 2 mg (1/30 grain) of atropine sulfate deep into the deltoid muscle, the heart began to beat normally no further syncopal attacks occurred and the patient made an uneventful recovery This observation is of great therapeutic interest and points to the action of the vagus in cases of heart block following rheumatic fever It agrees with the studies of Gross and Field who found minimal changes in the conduction system in rheumatic fever and concluded that vascular changes were more important than exudative Vagal release in these cases may increase the coronary flow

Poole and Wilkinson[303] have recently reported a case of complete heart block in a man of 72 where reversion to a normal rhythm occurred after administration of small doses of benzedrine sulfate (amphetamine sulfate) Twelve hours after administration of the first dose of 10 mg (1/6 grain) by mouth the pulse rose to 68 and there was improvement in the clinical status of the patient When the drug was withheld the block returned and it was again given with identical results The dosage of amphetamine was therefore maintained These authors believe that the drug is superior to epinephrine and ephedrine for this purpose Amphetamine is closely related chemically to both ephedrine and epinephrine and possesses similar pharmacologic properties, so this action in heart block is not unexpected

The treatment carried out in the patient under discussion was as follows When first seen a capsule containing theophylline ethylene diamine 0 12 Gm (2 grains) and phenobarbital 15 mg (¼ grain) was given after meals When evidence of congestive failure appeared, he was completely digitalized and then maintained on 0 1 Gm (1½ grains) of the whole leaf daily When the Adams Stokes attacks appeared the patient was given ephedrine hydrochloride in 20 mg (1/3 grain) doses every four hours by mouth Later a hypodermic injection of 30 mg (½ grain) was given every two hours We were afraid of epinephrine injections because of the nature of the arrhythmia seen in the electrocardiograms during the seizures

## Hypertensive Cardiovascular Disease Complicated by Complete Heart Block and Adams Stokes Seizures

**Case 80** Miss A M a retired school teacher of 76 was first seen on October 7 1938 following a "spell" of unconsciousness The patient had been in fair health until a week before when she suddenly became dizzy and lost consciousness During the seizure there were convulsive movements A similar attack occurred the day before admission to the hospital It was followed by vomiting and some sense of fullness in the chest

Physical Examination B P 168/78 Pulse 40 The heart was enlarged to percussion in all diameters the left border reaching 13 cm to the left of the mid sternal line The lungs were clear and the liver border was palpated two fingers breadth below the costal margin All reflexes were normal No edema was noted

Laboratory Data The blood Wassermann reaction was negative Blood count hemoglobin 7[illegible] per cent (Sahli); R B C 3 650 000; W B C 8 [illegible]00 Differential neutrophiles 64 per cent lymphocytes 36 per cent The urine showed specific gravity of 1 010 a heavy trace of albumin no sugar and a few hyaline casts Blood urea nitrogen

18 mg The roentgenogram of the chest showed the heart to be of the hypertensive shape with widening in all diameters

COURSE On the first day of a two weeks stay at the Woman s College Hospital this patient had a typical Adams Stokes seizure She gave warning of its approach during the taking of an electrocardiogram consequently the whole seizure was recorded (see Figs 15 and 216)

DISCUSSION Following the Adams Stokes seizure the patient was placed on barium chloride in 60 mg (1 grain) doses after meals There were no other attacks during her stay in the hospital Six months later a follow up note from her physician states that the medication had been continued with no recurrence of the seizures I do not believe however that her freedom from syncopal attacks came as a direct result of the therapy since there is always a tendency for these attacks to cease spontaneously They occur more frequently in some patients than in others for example in Case 79 the seizures were so frequent that they were not all recorded Usually the remedy that is administered at the time of the spontaneous recovery gains an undeserved reputation that it loses in a few years No specific drug for Adams Stokes seizures has yet been found and a multiplicity of remedies makes the therapy of this rare disease appear to the casual observer to be in a confused and unsettled state

Early experimental work showed that the salts of barium and calcium increased the irritability of cardiac muscle and in 1923 barium chloride was first used clinically Good reports appeared on all sides and its action combined with adrenalin caused favorable comment Starting with the report of Parsonnet and Hyman [94] who used the drug unsuccessfully in eight cases of complete heart block with Adams Stokes seizures the tide has recently turned against barium chloride

Lueth [40] has advised the use of metrazol in complete heart block with the Adams Stokes syndrome In four of his cases two were greatly benefited while in the other two it proved to be of little value However the drug has no untoward effects even when used over long periods a fact which recommends it when other measures fail Metrazol (cardiazol) is pentamethylene tetrazol a substance with camphor like action is soluble in water withstands heat and therefore can be safely and easily sterilized for subcutaneous injection The hypodermic injection varies with the patient Usually it is wise to start with small doses (½ to 1 cc of a 10 per cent solution) subcutaneously and at the same time to administer the drug by mouth Larger doses (5 to 7 cc of a 10 per cent solution) have recently been given intravenously in schizophrenia with few permanent effects on the heart In the Adams Stokes cases the beneficial action of the drug comes about through its stimulation of the vasomotor tone and respiration The same action recommends its use in cases of circulatory collapse

Thyroid U S P has been recommended for the prevention of Adams Stokes seizures in doses of ½ to 3 grains daily to increase the irritability and rate of the ventricles

It must be emphasized that complete heart block and Adams S

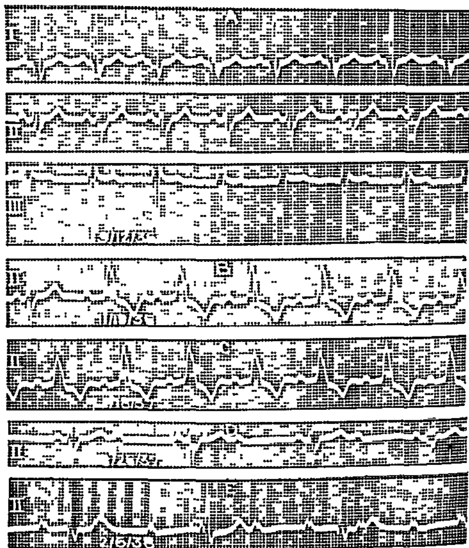


FIG 145 A series of electrocardiograms of a white male of 66 suffering from hypertensive cardiovascular disease and angina B P 10/100 heart moderately enlarged *when first seen On careful regulation of regime the clinical status showed no change* during the next three years The progress of his lesion is reflected in h electrocardiograms A Three standard lead taken at the time of his first examination Note marked left axis deviation and QRS B Lead _ from a tracing made two months later Note the transition from normal beat to bundle branch block There is also a slight prolongation of the P R Intervals No digitalis had been given C Two months later the bundle branch block persists D Seven months later A _ 1 heart block is present The bundle branch lesion is no longer in evidence E Six months later The heart block is now complete Tracings similar to this one were obtained over the course of the next two years The patient died in February 1940 six months after the onset of congestive cardiac failure No Adams-Stokes seizures appeared at any time

syndrome are not synonymous Heart block may be present for many years without the occurrence of faintness syncope or convulsive seizures This is well illustrated by the patient whose electrocardiogram appears in Fig 145

### Complete Heart Block Caused by Tumor Metastasis—Autopsy

**Case 81** C E F a white male laborer of 45 was admitted to the Philadelphia General Hospital complaining of swelling of the legs The onset was three years before admission when some edema appeared at night but the patient was able to work until 1/ 1/37 At this time he developed precordial pain increase in edema and cough

Physical examination showed B P 120/80 an emphysematous chest full of moist crepitant rales The liver was enlarged 3 cm below the costal margin There was no fluid in the abdomen The fingers showed slight clubbing and the ankles pitting edema moderate in degree The heart was enlarged to the left The apex beat was in the sixth interspace in the anterior axillary line and a soft systolic murmur was present over the apex No thrills Heart rate 60 to 70 and irregular

Laboratory Data Electrocardiogram showed right axis deviation and complete heart block The ventricular rate was more rapid than one would expect in heart block (60 to 90)

The roentgenogram showed cardiac enlargement and emphysematous lungs with diaphragmatic adhesions

Clinical Diagnosis A Etiologic Arteriosclerosis B Anatomic Cardiac enlargement C Physiologic Heart block Congestive failure D Functional Classification Class 3 Therapeutic Classification Class E

Course In spite of venesection digitalization and other measures directed toward the relief of the symptoms of cardiac failure the patient s course was rapidly downhill and he died two weeks after admission

Autopsy revealed about 200 cc of blood tinged fluid in the pericardial cavity The parietal pericardium showed no change but the visceral layer was covered with tumor nodules extending into the myocardium (Fig 146A) The left auricle and the right ventricle were filled with a mass of tumor tissue (Fig 146B) grayish red in color They were fairly firm not friable and were attached by pedicles to the region of the septum in places extending part way through the latter There was extensive tumor infiltration of the endocardium above the mitral ring but the valves were not involved in the neoplastic process The inferior vena cava was partially obstructed by large masses of tumor tissue in the right auricle The coronary arteries were unchanged

**Discussion** Metastatic tumor of the heart invading the septum and causing heart block is rare It must always be thought of however in patients with a primary tumor growth in any region of the body and circulatory symptoms of obscure etiology (Chapter 14) In this case the primary focus was thought to be a large ulcer in the stomach It was impossible to determine the exact type of tumor cell present either epithelial or lymphoid origin was suspected

## PULSUS ALTERNANS

### Hypertensive Cardiovascular Disease with Beginning Congestive Cardiac Failure—Pulsus Alternans

**Case 82** F H an Italian laborer of 64 was admitted to the Woman s College Hospital on 7/4 33 complaining of increasing dyspnea and edema He was examined in the cardiac clinic eight months prior to admission at which time the blood pressure was found to be 220/120




FIG 146 A Tumor nodules in epicardium B Tumor mass filling cavity of the right ventricle (Autopsy No 33 696 Philadelphia General Hospital)

Physical Examination The left cardiac border extended to the midaxillary line A systolic murmur was heard over the apex and there was dulness to the right scapular angle with the absence of tactile fremitus and breath sounds On admission the pulse rate as 1 0 with regular rhythm Half the pulse beats were found to come through at a blood pressure level of 200 at 190 the pulse rat suddenly doubled The diastolic level was 1?0 The weakness of the alternate beats was not appreciated on pulse palpation An electrocardiogram showed a left axis deviat on and an inversion of T 1

Clinical Diagnosis A Etiologic Hypertension B Anatomic Cardiac enlargement Relative mitral regurgitation C Physiologic Normal sinus rhythm Pulsus alternans D Functional Classification Class 4 Therapeutic Classification Class E

Discussion The detection of pulsus alternans in this patient confirmed the diagnosis of left ventricular failure and suggested a poor prognosis The treatment consisted of bed rest thoracentesis venesection and digitalization On this regime the patient improved and was edema free by the end of the first week in the hospital Two weeks after admission however he died suddenly during another seizure of paroxysmal no turnal dyspnea or sudden left ventricular failure Characteristic alteration of the pulse appeared at intervals during the last two weeks of life

Pulsus alternans can be demonstrated best by a graphic tracing of the radial pulse However whenever its presence is suspected an attempt should be made to demonstrate it by the use of the sphygmomanometer To do this the cuff is inflated above the systolic level of the blood pressure As the mercury column drops slowly only one half of the pulse beats will be heard coming through As the mercury falls still further (range about 5 to 15 mm ) a level will be reached where the pulse rate is suddenly doubled In other words all the pulse beats will be heard at this second systolic level

Alternation has been attributed to the fact that the myocardium as a whole may fail to respond to each contraction impulse because of the presence of advanced myocardial disease The refractory fibers are scattered through the whole heart and are sufficiently numerous in certain disease states for their absence to be detected when the strength of the pulse beat is recorded graphically The difference at times may be appreciated by careful palpation of the radial pulse Pulsus alternans may accompany hypertensive cardiovascular disease when there is beginning heart failure or it may appear following an acute coronary occlusion Where very rapid cardiac rates accompany this phenomenon the prognostic significance is not the same for a healthy heart in paroxysmal tachycardia or flutter may be so overloaded that rest of some of the fibers may be a protection mechanism necessary for survival The prolongation of the refractory periods of some fibers causes alternation when seizures are prolonged However in these instances pulsus alternans disappears as soon as the paroxysm is over Alternation in the height of the QRS complexes of the electrocardiogram is not identical with pulsus alternans When mechanical alternation is not present in the pulse tracing or demonstrable in the mercury column of the sphygmomanometer the electrocardiographic alternation is of no clinical significance

# 13

# FUNCTIONAL HEART DISEASE

C'est l'imagination qui gouverne le genre humain NAPOLEON BONAPARTE (*Bourrienne Life* II, 2)

Many patients have 'heart disease' that is wholly in their imagination Considering the percentage of the modern population who possess the constitutional background for the development of a neurosis of some type it is small wonder that present day radio and newspaper agitation concerning the increase of heart disease turns attention in this direction Not infrequently the presence of a harmless systolic murmur discovered quite by accident or the misinterpretation of an electrocardiogram or roentgenogram by a physician is the starting point of years of invalidism Many times the sudden death of a near relative from cardiac disease focuses the attention of the patient on the heart Likewise a person of the neurotic type who learns of the presence of organic heart disease may at once develop symptoms out of proportion to the lesion The symptoms of a cardiac neurosis will then be superimposed on those of a definite circulatory disease

On the other hand symptoms of heart disease often are present in patients who are not victims of fear or of psychoneurotic states They are frequently produced in individuals who are subjected to excessive exertion and loss of sleep over long periods of time Among the Union forces during the Civil War this syndrome was first recognized and described by Da Costa as the irritable heart of soldiers During the World War (1914 1918), Sir Thomas Lewis gave the name effort syndrome to the condition and recently American writers have suggested the more appropriate designation neurocirculatory asthenia" The last term is to be preferred since it does not contain any reference to the heart

## NEUROCIRCULATORY ASTHENIA

Neurocirculatory asthenia is a readily recognized syndrome characterized by palpitation dyspnea easily produced exhaustion and at times by chest pain following slight exertion While common in soldiers it is not infrequently met in civilian practice It is most essential to distinguish this condition whenever possible, from the psychoneuroses presenting cardiovascular symptoms Some cases however may be encountered that show a combination of the effort syndrome and the cardiac symptoms of a psychoneurotic nature

When a nation mobilizes, men from all walks of life and from all but

the vital occupations are gathered together and subjected to the stress and strain of army life Forced marches drilling varying degrees of mental trauma and loss of sleep are daily experiences If these are long continued circulatory symptoms develop sooner in some than in others Normally following exertion any untrained person shows breathlessness rapid heart action palpitation vertigo and faintness even precordial pain may accompany extreme effort Exhaustion and tremor often appear when activity ceases and if the oxygen debt is great breathlessness continues These same sensations occur in men subjected to army routine and cannot at the start be considered abnormal However as time goes on these symptoms are produced by less than ordinary amounts of exertion Consequently while the symptoms are not abnormal they are so easily produced we say the patient suffers from 'effort syndrome or neurocirculatory asthenia

Lewis found that the group invalided home from the British expeditionary forces in 1915 because of this disability was a mixed one [9] In it were included soldiers who showed no signs of actual heart disease but evidences of constitutional inferiority Many were of the hyposthenic type with long flat chests and family histories of psychoneurosis epilepsy or insanity Others were from divisions at the front line where activity had been great and the sojourn long Consequently exhaustion loss of sleep and continued strain were the precipitating factors In another group, the symptoms of neurocirculatory asthenia came on during convalescence from illnesses like tonsillitis influenza or pneumonia Others showed previously unrecognized incipient tuberculosis or other infections while a few showed beginning heart involvement All had the same symptoms but a wide range of etiologic factors Consequently the condition became very difficult to analyze in a truly scientific manner In the army treatment had to be prescribed for these patients as a class while in civil life individual and accordingly more successful management is possible

## Symptoms and Signs

**The breathlessness or dyspnea** of neurocirculatory asthenia is not present when the patient rests but appears very quickly when slight exertion is attempted It is probably of nervous origin since the vital capacity of this group is only a little below normal Excessive fatigue and exhaustion generally come on very quickly after slight exertion and are accompanied by tremor in many cases These symptoms closely resemble those produced by hypoglycemia following violent exercise Likewise pain over the region of the heart and even hyperesthesia of the adjacent skin surfaces occur on slight exertion The chest pain is an important symptom and must be differentiated from the pain of angina

**Palpitation Dyspnea and Fainting** Palpitation commonly attends slight exertion in effort syndrome and is generally caused by sinus tachycardia Arrhythmias are rare but extrasystoles occasionally are present Fainting occurs in this group of cases generally following slight trauma like the taking of blood from an arm vein It is usually vasovagal in origin

Examination of the heart shows little evidence of disease The only abnormalities will be the tachycardia and the increased forcefulness of the heart beat The cardiac rate is not uncommonly 85 per minute at rest, while higher rates are observed in patients who are ambulatory After exercise the pulse rates mount quickly in these cases and return very slowly to normal The blood pressure varies It is low in many patients at rest but tends to show an exaggerated response to exercise

### Incidence

Neurocirculatory asthenia is not uncommon in civilian practice The stress and strain of life overwork exhaustion and worry bring on the syndrome in many who have normal hearts Women have been found to predominate in the cases studied by Edwards and White[80] In a series of 5000 consecutive private cases these observers report a definite diagnosis of neurocirculatory asthenia in 13 7 per cent The majority of their patients (85 8 per cent) were under 50 years of age

Twenty five years ago at the outbreak of the first World War the symptoms observed in the effort syndrome group were thought to represent the early warnings of various disease states However the years have passed and hypertension and hyperthyroidism have not appeared in excess of the expected ratio in the groups that have been followed With the world again at war the number of cases will increase because the provocative factors of fear hunger and exhaustion are certain to reappear Recurrence of the symptoms in the same persons is not unlikely since the basic constitutional factors producing them still exist

Neurocirculatory asthenia occurs more often in individuals with perfectly normal hearts (65 2 per cent of White s series) but there is nothing to prevent its occurrence in those with heart disease (19 6 per cent of White s series) who possess unstable neurotic personalities In the latter group the signs of an organic cardiac lesion will be recognized but the examiner will be struck at once by the exaggerated character of the symptoms when viewed in the light of the amount of demonstrable cardiac damage Rheumatic heart disease is the type most likely to be complicated by neurocirculatory asthenia

### Management

The management of the patient with true neurocirculatory asthenia is by no means easy However the earlier the condition is detected in army practice and proper treatment begun the quicker will the soldier be able to return to full duty Removal from the front line is usually in itself sufficient to produce some degree of improvement particularly if rest is the first measure employed Tension is relieved better hygienic surroundings prevail, and these factors at once favor convalescence

The first essential in treatment is to give the patient an honest statement of the exact status of his case This takes time He must be shown

the result of the thorough physical examination including electrocardiogram and orthodiagram, and its significance explained He must be told that there is no evidence of heart disease, but that the annoying and incapacitating symptoms have appeared because he has shown a lack of training Health he must be made to realize will be regained by a process of training and exercise is in order not bed rest The type and duration should fit each case (Chapter 20) Individual care while not possible in the army where the exercises are carried out in groups is possible in civilian practice and leads to a quicker result than mass handling

**Regime** The patient s day should be planned with care and the program written out during the consultation No deviations should be allowed The number of hours of rest at night should be specified coffee tea tobacco and alcohol forbidden and the strenuous features of the daily work at first prohibited The patient must realize the importance of starting treatment on a much curtailed schedule even though this attracts the attention of coworkers to him At all costs he must be launched on an exercise allowance that he is capable of maintaining and this can be gradually increased according to the response shown at subsequent visits Exercise is always prescribed in a precise manner and amounts raised as tolerance is acquired

**The general physical examination** may reveal some foci of infection in teeth tonsils sinuses or prostate These should be evaluated (page 440) and any other abnormality present should receive appropriate attention Tuberculous infection calls for special management

**Use of Drugs** As far as compatible with good practice drugs should be avoided but if needed in the treatment of neurocirculatory asthenia they should be directed toward the minor complicating symptoms like headache constipation etc As far as the condition of neurocirculatory asthenia itself is concerned drugs are of little value The patient should be made to realize the cause of his condition then the fewer drugs used in the treatment the better Digitalis accentuates the symptoms particularly the palpitation and should not be used If secondary anemia is found iron and the usual dietary measures may be prescribed at the start of treatment

**Re examination** of the patient at intervals is justified to make sure that the diagnosis originally advanced remains correct Heart size following additions to the exercise prescription should be determined orthodiagraphically and the retrocardiac space viewed on each occasion to rule out the presence of mitral stenosis which is often suspected because of the accentuated character of the first heart sound On the occa ion of one of the follow up visits a basal metabolism determination should be made to eliminate hyperthyroidism as a contributing factor Routine observations of the blood pressure temperature weight and cardiac rate are likewise valuable in directing treatment

Throughout the entire course of therapy the greatest care should be taken to avoid either saying or hinting that any of the symptoms that the

patient describes are imaginary Be prepared to discuss each one at length at any visit If the practitioner does not have time to do this, he should refer the patient for the proper treatment when the diagnosis is established Discouraged and neglected patients generally take their problems and they are real ones to irregular practitioners

### Prevention

Prevention of neurocirculatory asthenia at least of the more severe grades is quite possible during war time if recruits in training camps are efficiently graded upon their arrival Estimation of the capacity to stand certain forms of activity could be ascertained by a schedule of graded exercise prescribed for each new group This sorting-out process is impossible on the basis of physical findings alone although certain clues, previously mentioned are obtained at the first routine examination Lewis [9] has listed some of the graded drills useful in these cases others may be found in Army drill manuals

## ILLUSTRATIVE CASES

### Neurocirculatory Asthenia in the Absence of Signs of Heart Disease

**Case 83** A T an unmarried music teacher of 40 was first examined in October 1937 when she complained of palpitation rapid beating of the heart and dyspnea on moderate exertion She was nervous and apprehensive as a child and avoided activities while at boarding school spending most of her spare time studying music She could not stand much excitement or overexertion Her father died suddenly from what was described to her as heart failure Since his investments had been poor she was obliged following his death to make her own living teaching music She began to worry about finances and lost much sleep She became conscious of her heart action about this time and noticed increased dyspnea and palpitation when walking up the steps to her studio Her pulse would mount to 110 1 0 on climbing one flight of stairs

Examination showed a normal blood pressure measuring 110 systolic and 80 diastolic There was no cardiac enlargement (the orthodiagram showed a small heart of ptotic type) There were no murmurs and the rhythm of the heart was rapid but regular The electrocardiogram blood count and urinalysis were normal

**Discussion** In the first place the background of this patient is suggestive Her school history shows that she was of the frail type inclined to spend more time in the study of music than in developing normal social contacts She was an only child Consequently we should not wonder at the type and severity of the reactions after her father s sudden death Worry loss of sleep and the increase in her activities made necessary by the fact she had to make her own living in a studio some distance from her home precipitated the symptoms of neurocirculatory asthenia She was convinced she had always had a weak heart and the appearance of these marked symptoms on exertion firmly fixed this belief in her mind

Treatment was begun by telling the patient the symptoms she had experienced were indeed real and followed a praiseworthy attempt to make her way against great odds The amount of exertion she had been forced

to expend to open a studio and to renew contacts with many friends in the hope of acquiring pupils was far above her accustomed level Consequently an untrained circulatory system was giving her notice of neglect of exercise that had attended long years at the piano

The result of the examination of the heart was explained in detail and the normal roentgenogram and electrocardiogram were offered as additional evidence She was advised to begin her work on a smaller scale in a studio on the street level Out-of-door exercise in the form of graded walks was prescribed She was encouraged to seek relief from worry in a different home environment where she would not be alone in the evening

No cardiac medication was prescribed A sodium bromide mixture was given for the first two weeks By the end of this time definite improvement was reported The patient was seen at long intervals after this and she stated that all cardiac symptoms were less evident and had ceased to annoy her at all when she was working in the company of friends or attending concerts Considering this patient s build and disposition the background for the reappearance of the symptoms of effort syndrome is ever present so we cannot say she is cured Insomnia overwork or worry might cause their return at any time

### Neurocirculatory Asthenia—Increase in Symptoms Under Medical Management—Relief Following Adrenal Denervation (Presented by Dr James Lehman)

**Case 84** Mr J M age 23 was admitted to the Woman s College Hospital complaining of 'rapid heart beat for the past eight months At times the rate was so rapid that it was impossible to count Precordial pain on slight exertion was added to the picture a month later and he was forced to give up his work as a grocery clerk Upon advice of a physician he went to bed for three weeks and took digitalis He was unimproved After this he was given iodine for a period of two months He gained some weight (six pounds) during this time but felt much worse He was weak, became exhausted on slight exertion and the chest pain and rapidity of cardiac action were still present

Examination revealed a well nourished and well developed adult male who was intelligent and highly co operative The pupils were equal moderately dilated and reacted to light and in accommodation There was no exophthalmos The thyroid was slightly enlarged The lungs were normal The heart was not enlarged and there were no murmurs The rhythm was regular and the rate varied from 70 to 1[illegible]2 per minute B P was 136/86

Laboratory Data Several urinalyses were normal Hemoglobin was 90 per cent (Sahli) R B C 4 870 000 W B C 6 300 polymorphonuclears 60 per cent lymphocytes 37 per cent monocytes [illegible] per cent eosinophiles 1 per cent Blood urea was 16 *glucose 100 basal metabolic rate* was plus four

Clinical Diagnosis A Etiologic Effort syndrome (neurocirculatory asthenia) B Anatomic No structural disease C Physiologic Normal sinus rhythm D Functional Classification Class I

**Discussion** This young man showed a clinical picture that is typical of neurocirculatory asthenia He was tall and slender and there was marked lordosis The response to slight exertion when he was first seen was exaggerated and consisted of tachycardia breathlessness and chest

pain Great variability of the heart rate was present at all times, a mere change in body position was often sufficient to cause an elevation of the pulse rate of 50 beats a minute Associated with this were tremor shakiness dizziness sweating and excessive fatigue We note that a determination of the blood sugar was normal The fatigue on exertion was following by such a marked feeling of exhaustion that he was compelled to give up his job and go to bed at which time all symptoms became much worse Digitalis was certainly not indicated and it is not surprising that no improvement followed its administration

It is interesting to note that iodine was then given a therapeutic trial no doubt with the thought in mind that the condition might in spite of the normal basal metabolic rate be due to overactivity of the thyroid gland There was a slight gain in weight but the symptoms remained the same

Many times neurocirculatory asthenia may appear to simulate thyro toxicosis but closer inspection of the patient often reveals wide differences It is very noticeable at the start that the patient with hyperthyroidism is optimistic and complains very little while the patient with neurocircula tory asthenia is discouraged and constantly complains of many vague symptoms The patient with thyroid overactivity is bold ambitious alert, and moves quickly while the patient with neurocirculatory asthenia is con stantly exhausted and moves slowly The appetite is normal or increased in thyroid disease while in effort syndrome, it is poor The skin in hyper thyroidism is warm moist and pink and of unusually fine texture while in neurocirculatory asthenia cold moist (often cyanotic) extremities are seen Even the tachycardia differs in the two conditions When caused by thyroid overaction it is persistent and sustained while in neurocirculatory asthenia it is variable

After many years of research Crile [6 78] has concluded that neuro-circulatory asthenia is a pathologic state in which there is an excessive stimulation of the adrenal sympathetic system He has attempted to reduce this adrenal overactivity by denervation of these glands

In our experience adrenal denervation combined with denervation of the aortic plexus or resection of the splanchnic nerves has given *gratifying* results in a small carefully selected series of cases The operation is attended by little risk and to date there has been no mortality

This patient showed no result following prolonged medical treatment, consequently on 3/31/36 a left adrenal denervation and splanchnic resec tion were performed The right side was done 4/11/36 The convalescence was smooth except for some abdominal distention The patient was dis charged 14 days after the second operation

There was immediate improvement in the heart action this was followed by a gain in weight and strength Later the patient returned to school at the same time working extra hours in the grocery store

On 12/29/36 a gain of 15 pounds was reported The pulse was 76 B P 130/90 At the time of this visit he was attending school and working during week ends There was no recurrence of rapid heart action

1/28/37 The patient reported a gain of three additional pounds The pulse was 80 B P 130/80 No symptoms

On 5/13/39 the patient stated that he was working every day in an office There were no symptoms Activities had been gradually increased in fact he was able to play tennis occasionally

## PSYCHONEUROSIS

The beginning of health is to know the disease CERVANTES
*Don Quixote* Pt 11 Ch 60

Symptoms referable to the cardiac mechanism may develop in psychoneuroses of any type Those most frequently involved are the fatigue neuroses (neurasthenia) and the introspective neuroses (hypochondria) Anxiety states have cardiovascular symptoms which are apt to be built around chest pain or cardiac palpitation Substitution neuroses (hysteria) or obsession states (psychasthenia) less often present cardiac problems

### ETIOLOGY

Psychoneurotic states occur in patients with established heart disease as well as in normal individuals The remark of a medical examiner concerning the presence of a heart murmur may first direct attention to the heart The adventitious sound in question may be and indeed often is entirely functional yet fixation is established and the patient is started on the road to invalidism The road back if there is one is long and difficult It is just as grave an error to diagnose heart disease on the basis of a functional murmur as it is to miss the diagnosis of an early organic lesion Hasty decisions rendered in dispensary or office in regard to cardiac disease are unfortunately at the bottom of many of the cardiac psychoneuroses Although the patient s story may sound typical of neurosis a careful and complete cardiac study should be made before an opinion is rendered Snap judgment is always dangerous

The present-day tendency on the part of newspapers to acquaint the public with many facts concerning heart disease commendable as it may be is not without serious drawbacks Too much attention often is paid to symptoms of little importance by a large group of potential psychoneurotics who are unable to sort the wheat from the chaff The health talk many times serves to establish firmly the idea of heart trouble because the description always seems to fit The sudden death of a near relative from heart disease may kindle fear and start the neurotic on the rounds of physicians offices Pain caused by any thoracic or abdominal disorder and referred to the upper abdomen or chest may be the innocent cause of much needless alarm Given the proper soil a host of incidents is capable of establishing fixation on the cardiac apparatus Often symptoms having their origin outside the heart may be responsible Since the heart of all organs in the body commands the most attention a fear of sudden death

arises when pain in the chest or any abnormality of cardiac rate or rhythm probably in themselves innocuous force their way into conscious perception The fear of heart disease becomes deep rooted and is difficult to dislodge

## Diagnosis

It is always well to allow the patient with a functional heart ailment to tell his own story in his own way without interruption This privilege will contribute greatly to the diagnosis since the psychoneurotic individual usually will describe a multiplicity of symptoms If there is fixation on chest pain the story the patient presents may often be the perfect description of angina acquired as a result of much travel in medical circles However when questioned there will generally be some small detail of the account that will not fit the picture the relationship of the pain to exertion its radiation or the presence of precordial tenderness

**Palpitation** is a common cardiac complaint among psychoneurotics sometimes dating back to a scare in childhood The fear produced by the original accident is at once recalled whenever palpitation occurs Soon the patient becomes acutely conscious of the heart action and is quite convinced that serious organic heart disease is present Advice delivered over the radio at this point is no help The occurrence of premature beats or extrasystoles while harmless increases the introspection and the psychoneurosis becomes more deeply rooted These patients may describe every skip of the heart for a 24 hour period (Case 69) a symptom in itself quite suggestive of psychoneurosis

**Other Symptoms** While precordial pain tachycardia, and palpitation are most commonly complained of any of the symptoms of cardiac disease may be presented as the chief complaint by a psychoneurotic individual Dyspnea even the nocturnal variety may be described in addition to fainting vertigo coldness of hands and feet and a sense of suffocation A thorough deliberate physical examination at the start impresses the patient and will satisfy the examiner that organic heart disease is absent It is important to avoid haste in this initial study in order that the slightest detail will not be omitted for the average psychoneurotic patient is well informed concerning the procedures to be expected as part of a modern cardiovascular examination During the course of auscultation the physician is closely watched consequently it is wise not to pause too long in any area If a systolic apical murmur is present in a rapid overactive heart which often accompanies the psychoneurotic state, it is wise not to pay too much attention to it If the patient has previously been informed of its presence he will usually tell the physician when this stage of the examination is reached It is best to defer answering questions concerning any of the signs elicited until the entire study has been finished The importance of the systolic murmur can then be stated in the summary

**Successful treatment** of cardiac symptoms that are a part of a psychoneurosis is difficult and many times it will require the skilled services of a

psychiatrist If the physician undertakes to treat one of these patients he must realize at the start that it is a time consuming procedure and if he does not have sufficient time and interest he should refer the patient to another physician

At the initial interview, the patient should be encouraged to tell his own story Much can be accomplished in an atmosphere where hurry is not evident if a sympathetic and encouraging attitude is adopted by the physician Otherwise he is not likely to hear the whole story and pertinent details may be withheld Let the patient have his say Make no attempt to have the story related so as to follow a convenient form Such a history may be useful for filing but in the end it generally contains a conglomeration of facts that lead nowhere In addition many patients will hesitate to relate the whole story of an illness when they notice that every thing is being taken down by the physician

When the history and physical examination are finished the physician should tell the patient that he has cardiac symptoms but that the cause is evident and complete cure is possible It is obvious that a definite statement like this cannot be effective unless the full confidence of the patient has been gained by a careful and painstaking examination

Usually a search through the data will reveal the cause of the heart attacks in some emotional incident Emphasizing this association and giving the patient the necessary encouragement successfully launches the cure Similar incidents may then less readily provoke symptoms Improvement may be expected to follow in a few weeks In all doubtful cases and in those where response is not evident in a reasonable time the opinion of a psychiatrist should be obtained

## ILLUSTRATIVE CASES

### Cardiac Psychoneurosis Complicating Rheumatic Heart Disease

**Case 85** Miss A M a single American clerk of 25 was first seen in June 1936 There was no previous rheumatic history or illness of any nature On insurance examination the month before a heart murmur was discovered and the patient was informed that she had heart trouble Since then she developed dyspnea palpitation weakness vertigo insomnia and a catch in her breathing (sighing respiration?) She began to miss many days at work and when examined was contemplating resigning her position She expressed the fear of sudden death

Physical Examination showed a blood pressure of 120/80 very slight cardiac enlargement a presystolic murmur and accentuated first sound over the region of the apex The electrocardiogram showed right axis deviation and notched P waves in leads and 3 An orthodiagram showed mitralization and slight congestion in the hilar regions on both sides There were present superficial and deep tenderness in the left precordial area

Clinical Diagnosis A Etiologic Rheumatic Psychoneurosis B Anatomic Cardiac enlargement Mitral stenosis Mitral insufficiency C Physiologic Normal sinus rhythm D Functional Classification Class 1

Discussion There is no doubt that this girl had a well established mitral stenosis unrecognized until the time of the insurance examination

When this fact was communicated to her she at once associated it with *sudden death* She was a hypersensitive nervous individual already possessing the mechanism necessary for the development of a psychoneurosis The statement of the insurance examiner was the precipitating factor Some physicians are of the opinion that the diagnosis of psychoneurosis should be advanced only when the physical examination and laboratory studies are entirely negative However, this is far from the case in many instances as this patient shows

The physician who examined this girl mentioned the heart murmur and advised her to consult her own physician To be sure his advice would be followed he laid too much emphasis on the gravity of the situation as a whole

At the start of the treatment of an uncomplicated psychoneurosis an explanation of the symptoms experienced by the patient is easy, but the task becomes more difficult when there is a background of organic heart disease It should however be attempted The care necessary for the existing lesion should also be stated and the good reserve strength of the heart muscle demonstrated Most of these patients when first seen have been given digitalis some in large doses The withdrawal of this drug may be stressed to drive home the statement that the heart muscle although damaged has sufficient reserve which can be retained for many years

Schnur[331] has recently suggested as a therapeutic test for differentiating the symptoms of cardiac psychoneurosis from those of organic heart disease the injection of novocaine intradermally at the affected site The hyperalgesia tenderness and pain disappear at once Often the symptoms of the psychoneurotic state will be relieved by this procedure Pain due to organic heart disease will not be relieved by this method

Harmless arrhythmias many times may precipitate a cardiac neurosis In instances where the arrhythmia appears following trauma it may continue to produce symptoms in spite of all therapeutic effort until a legal settlement has been made The patient whose history appears in Case 69 showed improvement following small doses of quinidine sulfate, a sedative mixture and increase in the prescription of out of door exercise

In some patients over 40 years of age the precordial pain that attends frequently recurring premature beats may be misinterpreted as the anginal type The patient may hear of this in which event it serves to augment the symptoms of the psychoneurosis

These patients should be completely examined at the start and all tests made A large dose of reassurance should then be administered and at subsequent visits the patient should be advised and educated Repetition of an entire physical examination or special examinations in these cases at too frequent intervals serves to make the patient suspect he was not told the truth at first Opinions rendered must be positive to be of value Advice at the end of the interview 'to take things easy wastes the time spent before this remark for the patient usually cannot link this advice with the previous statement that he has a perfectly normal heart

## Cardiac Psychoneurosis During the Course of Coronary Disease

**Case 86** G C a Hebrew tailor of 54 as first seen in January 1934 Three months before he had an attack of sever chest pain which as proved on electrocardiographic study to be caused by a posterior coronary occlusion He remained in good condition clinically and the laboratory stud es pointed to progress in the healing of the area of infarction However his complaints ere multiple pain in the left shoulder recurring transient pains over the precordium insomnia vert go epigastric and substernal burning and fear of sudden death

Physical examination showed B P 1 0/80 no cardiac enlargement a soft systolic apical murmur Ther were areas of superficial and d ep tenderness over the precordium

Clinical Diagnosis A Etiologic Arteriosclerosis Psychoneurosis B Anatomic No cardiac enlargement Coronary cleros s Cardiac nfarction C Physiologic Anginal syndrome D Functional Classification Class 3 Therapeutic Classification C

**Discussion** The coronary occlusion affected an area of cardiac muscle in this patient producing an infarction which healed satisfactorily This event had a marked effect on the nervous system precipitating a typical psychoneurosis which was the cause of the prolongation of the symptoms In the presence of a serious organic lesion of this nature many physicians are hesitant in considering any of the symptoms that may be subsequently presented as functional when the initial findings point conclusively to a serious heart disorder However all the pains in the chest complained of by this patient could not have been due to small occlusions At least frequent electrocardiograms did not show them Neither could we assume that these atypical pains were anginal They did not respond to nitroglycerine on all occasions and associated with them we had to consider the man s personality race and the presence of areas of hyperalgesia and deep tenderness over the precordium

The first problem here was to discuss the situation frankly with the patient and his family and write down a graded system of increased activities with the aim of dislodging the patient from bed where he had been invalided for too long a period His pains were not made worse by the end of the first week on the new program Consequently his allowance was increased during the second week At the end of this period his condition continued to improve so he was allowed to return to his shop in a supervisory capacity beside the cash register After this his complaints were few His medication consisted of a capsule containing 15 mg (¼ grain) of phenobarbital and 0 2 Gm (3 grains) of theophylline ethylene diamine after meals

## Cardiac Psychoneurosis and Congenital Heart Disease

**Case 87** Mrs C A a housе f of 30 as first seen in D ce nber 1933 when she compla ned of precordial d stress eakness vertigo and insomnia She was emotionally unst ble wept easily an l pr f ely at the slightest provocation The family history was sugge tive Th only ch ld a d ughter of ten as an i nbec le The patient stated that she had always been eak because of heart disease at birth She as not a "blue baby but the f m ly phys an told her mother that she had a leaking heart

Physical examination sho ed sl ght car i ac enl rgement a systol c thrill in the third left inter pace accompan ed by a h gh pitch d systolic n urmur The fingers re

not clubbed and no other defects were noted on physical examination The electrocardiogram showed high voltage of the QRS groups

**Discussion** Cardiac psychoneurosis when associated with organic heart disease will usually be found complicating coronary, rheumatic or hypertensive types since these are the most prevalent It is rarely seen combined with congenital heart disease because of the infrequency of congenital defects in the cases seen in practice

This patient was a typical psychoneurotic individual The idea of heart disease became firmly fixed in her mind at an early age In consequence she always led a sheltered secluded life at school and at home Her mother presented a similar problem in neurosis and was treated for the combination which causes most practitioners to seek shelter essential hypertension chronic arthritis and mucous colitis The patient took little or no exercise She was carried up the stairs as a child and always lived in a first floor apartment during married life Obesity developed in later years and thus accentuated the dyspnea that appeared on slight exertion The psychoneurosis was so deep-seated that the usual measures over a considerable period were ineffectual in bringing any measure of relief The signs of her lesion were so typical and the family history appearance and actions so characteristic of the superimposed psychoneurotic state that this patient was used for clinic demonstration on frequent occasions The last time she was shown the complaints of weakness malaise and slight fever failed to attract the attention of the clinician When the patient failed to reappear for the next examination our social worker traced her to the ward of another hospital The diagnosis of subacute bacterial endocarditis had been established by positive blood culture *

The lesson to be learned here is never to neglect to follow up the new symptoms that appear even though the diagnosis of psychoneurosi has been previously established It is also important not to overlook the possibility of the more common complications that attend the cardiac lesion

* Autopsy findings Large (uncomplicated) interventricular septal d fect contain n the vegetations typical of subacute bacterial endocarditis (Courtesy of Dr Geor e C Griffith )

# 14

# MISCELLANEOUS TYPES

Up to this point we have considered the main features and the management of various etiologic types of heart disease (page 60) The majority of the remaining conditions will be rarely encountered by the practitioner and our present knowledge of the other types is still far from complete Consequently brief descriptions of this miscellaneous group that includes the influence of anemia neoplasms pulmonary disease hypotension other infections and toxic agents on the heart although entirely unrelated will be grouped for convenience in this chapter Since most of the cases where cardiac trauma is suspected are seen with the surgeon the discussion of this subject has been placed in Chapter 17

## ANEMIA

In the presence of severe anemia any changes observed in the heart on physical examination should be interpreted with care for when the hemoglobin falls below 50 per cent symptoms closely simulating heart disease may appear The circulation time is decreased in anemia while the cardiac output and the minute volume show an increase These changes may be regarded as safety mechanisms to bring the amount of oxygen transported to the tissues nearer the normal level

The heart muscle in anemia is flabby and shows the familiar tiger markings chiefly in the left ventricle (Fig 147) Of 23 cases of Addisonian anemia reported by Cabot[49] hypertrophy and dilatation were observed at necropsy in 22 which agrees with other reports that an increase in heart size is present in advanced anemic states (chlorosis secondary anemia primary pernicious anemia) Cardiac enlargement has also been observed following the experimental production of anemia in animals

### Symptoms

The symptoms induced by anemia are breathlessness vertigo palpitation weakness and at times anginal pain on exertion Likewise a lessening of the blood supply to the legs particularly if some degree of sclerosis already exists may produce intermittent claudication The cause of both types of pain is muscle anoxemia Cabot reported three cases of typical angina associated with pernicious anemia in patients who at autopsy showed no coronary change In each instance the pain was produced by exertion and relieved by rest If advanced coronary disease is absent complete relief of the angina is possible in these cases following intensive treatment of the anemia and improvement in the blood count

Signs

In advanced anemia, cardiac enlargement may be evident on percussion The heart sounds are usually weak Systolic murmurs are often present in the region of the apex and at the base and their quality differs in no respect from those murmurs heard in patients who have actual valvular disease The murmur at the apex is considered to be secondary to a relative dilatation of the mitral ring with the production of mitral regurgitation

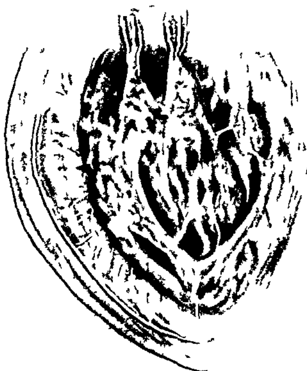


Fig 147 Tiger mottling in anemia

Dilatation of the aorta and pulmonary artery may be accompanied by murmurs in these areas In severe cases diastolic murmurs have been described Consequently considerable care must be taken in anemic individuals to avoid making the diagnosis of heart disease since the murmurs of anemia may simulate mitral stenosis Advanced untreated anemia may ultimately cause congestive manifestations as Cabot s series of autopsied cases have demonstrated However, where breaks in compensation have occurred pre existing heart disease of another type has usually been the cause, the anemia merely adding the final load that precipitates cardiac failure

When asphyxial blood is perfused through the coronary arteries it produces changes in the electrocardiogram similar to those seen when a coronary vessel is clamped Such evidence suggests that the changes brought about by anemia may be confused with those that arise secondary to a coronary occlusion In these instances it has been suggested that the change in the RS T interval in anemia is caused by a high concentration of locally produced metabolites

### Management

When the anemia is controlled all cardiac signs and symptoms may disappear The heart size if determined by accurate methods will be found to be smaller The various murmurs may no longer be heard and the anginal pain is no longer experienced Even in the presence of coronary disease improvement in the blood picture may often sufficiently raise the threshold to pain on ordinary exertion Re examination of a number of patients efficiently treated for progressive pernicious anemia after the blood count is normal reveals only the signs of cardiovascular disease that *one would expect to encounter in any similar group of patients at the same* age period

## PULMONARY HEART DISEASE
## (Cor Pulmonale)

Increase of pressure in the systemic circulation is followed sooner or later by hypertrophy of the left ventricle and hypertensive heart disease follows If the pressure in the pulmonary circulation is increased either suddenly by a large pulmonary embolism or failure of the left ventricle or more gradually by mitral disease or chronic pulmonary fibrosis we speak of the resulting condition as cor pulmonale or pulmonary heart disease

### Acute Cor Pulmonale

#### ETIOLOGY

The sudden right ventricular strain and dilatation that follows a large pulmonary embolus has been designated acute cor pulmonale by McGinn and White [46] Death in Case 29 was hastened by the rupture of an aortic aneurysm into the pulmonary artery and the sudden increase in the pressure in the lesser circulation Venous thrombosis following operative procedures has been shown to be the most common source of the emboli causing acute cor pulmonale

#### SYMPTOMS AND SIGNS

The production of acute cor pulmonale requires the blocking of at least 60 per cent of the pulmonary arterial circulation The symptoms are sudden in onset and consist of severe chest pain and dyspnea these are

usually quickly followed by signs of shock Death very often occurs suddenly after a massive occlusion of one of the main branches of the pulmonary artery while after a less extensive obstruction, survival may be attended by signs of right sided cardiac failure Cardiac size may increase particularly to the right, and venous engorgement may appear Electrocardiographic alterations resembling those produced by acute coronary occlusion may be observed but unlike those produced by coronary accidents *the changes brought about by pulmonary embolism* usually fade in a few days as balance is restored (see Fig 251)

The management of acute cor pulmonale has been included under the cardiac emergencies (page 509)

## Chronic Cor Pulmonale

The gradual replacement of lung areas by scar tissue and the obliteration of many branches of the pulmonary artery produces in some cases an interference with the blood flow Consequently a strain is placed upon the right ventricle and in time hypertrophy develops and later a right sided heart failure may appear

### ETIOLOGY

Chronic cor pulmonale may be secondary to a number of diseases of the lung Emphysema tuberculosis and pneumoconiosis commonly produce it while primary pulmonary endarteritis obliterans (Ayerza s disease) a rare condition is invariably accompanied by the signs of chronic pulmonary heart disease Chest deformities may place a strain on the right ventricle while metastatic carcinomatous infiltration of pulmonary arterioles may produce the same effect in some cases

*Chronic cor pulmonale may develop at any age Since the* lesions most apt to initiate the process occur in older people, most of the instances encountered clinically fall in these groups Primary pulmonary arteritis however often occurs in younger patients

Enlargement of the right ventricle dilatation of the pulmonary artery, and an increase in size of the right auricle are the main pathologic changes that occur in the heart Elevated pressure in the pulmonary circuit may also lead to atheroma of these vessels This condition however must not be confused with the change that takes place in the endothelium in cases of pulmonary endarteritis obliterans

### SYMPTOMS

The symptoms of chronic cor pulmonale are those of the underlying pulmonary disease in addition to the symptoms that result from strain and failure of the right side of the heart Dyspnea cyanosis hemoptysis clubbing of the fingers and toes may be secondary to the pulmonary disease Polycythemia often appears The signs and symptoms that direct attention to the heart are venous engorgement, the typical cardiac silhouette (see Fig 148A) the accentuation of the pulmonic second sound

and at times a pulmonic systolic murmur Extreme cyanosis ("black cardiacs ) accompanies advanced degrees of pulmonary sclerosis that may occur in some cases of chronic cor pulmonale Arrhythmias do not very often complicate the picture

### DIAGNOSIS

A bedside demonstration of right ventricular enlargement is difficult in these patients owing to the low diaphragm and the emphysema The right ventricle moreover faces anteriorly and changes in its size tend to produce alterations that are not appreciated by percussion The electrocardiogram in pulmonary heart disease shows a right axis deviation (see Fig 148B) and for this reason is valuable in diagnosis

The problem of differentiating between symptoms of pulmonary origin and those caused by cardiac insufficiency often arises Additional laboratory tests may be valuable in helping the clinician out of this difficulty The most useful of these are the measurement of the venous pressure and the arm to lung time (ether method) and the lung to tongue time (decholin method minus the ether time) [87] Abnormal circulatory measurements point to myocardial failure

In contrast to acute cor pulmonale the chronic form may show a slow progression over a number of years Resistance to pneumonia is generally poor consequently this disease takes a heavy toll particularly among old people Circulatory failure is also a common cause of death and is apt to follow a chronic course The prognosis in most cases depends upon the nature of the underlying pulmonary or cardiac lesion

### MANAGEMENT

The general plan of management is based upon the nature of the disturbance giving rise to the cor pulmonale When heart failure appears the usual measures are employed including rest digitalis and diuretics (page 74) If cyanosis deepens oxygen is useful (page 96)

A further discussion of the effect of asthma on the heart and the treatment of asthma complicating heart disease will be found in Chapter 16

## ILLUSTRATIVE CASE

### Chronic Pulmonary Heart Disease (Cor Pulmonale) Secondary to Bronchial Asthma of Long Duration

**Case 88** E W a salesman of 43 was first seen in August 1938 complaining of shortness of breath and cough Frequent attacks of asthma following respiratory infections had been present since childhood Cough was continuous for the past ten years increasing in severity during the winter Dyspnea was marked

Physical examination showed cyanosis and emphysema The heart sounds were distant and regular Rate 100 No murmurs were heard B P 110/80 The liver was palpable and tender a few centimeters below the right costal margin Slight ankle edema

Laboratory Data The electrocardiogram showed a right axis deviation (Fig 148B) The roentgenogram showed cardiac enlargement and an increase in the region of the

pulmonary conus (Fig 148 A) There was al o enlargement in the region of the right ventricle The Wassermann was negative The blood count showed R B C 5 700 000 and W B C 9 00

CLINICAL DIAGNOSIS A Etiologic Chronic cor pulmonale secondary to bronchial asthma B Anatomic Cardiac enlargement C Physiologic Normal sinus rhythm



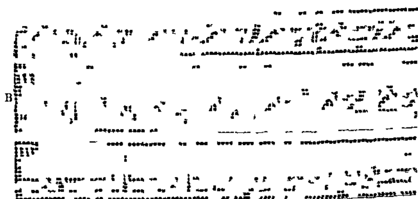




FIG 148 A Roentgenogram Note dilatation of pulmonic conus B The electrocardio gram Note presence of right axis deviation

Early (right si l d) cardiac failure D Functional Classification Class 3 Therapeutic Classification Class C

**Discussion** The history of attacks of asthma for many years and the marked emphysema discovered on physical examination in this patient furnish the necessary background for the diagnosis of chronic cor pul

monale The presence of right sided heart failure was indicated by the cyanosis engorged jugulars large liver and peripheral edema The roentgenogram and the electrocardiogram completed the picture

The precipitating cause of the failure was not evident from the history It may have followed the most recent upper respiratory infection Treatment was directed toward the cardiac failure and bed rest and digitalis were prescribed Digitalization was accomplished in one week but the cough continued Ammonium chloride in 1 0 Gm (15 grains) doses was given every four hours and codeine sulfate 30 mg (½ grain) was useful on a few occasions at night During the second week much relief was obtained from two intravenous injections of mercupurin (2 cc ) at five day intervals although at the time they were given no edema was evident

The patient was allowed to be out of bed at the end of the second week Digitalis maintenance dosage was continued and at the beginning of the third week he was referred for study of his bronchial asthma (page 462) Since he was local agent for a steamship company he was advised to seek transfer to a branch office in the south where better climatic conditions were known to prevail

## TUMORS

Tumors of the heart and pericardium are very rarely the cause of cardiovascular symptoms All too often they are discovered by the pathologist when the possibility of their presence was not even considered by the clinician On reviewing the rather extensive literature on the subject we note that every kind of tumor may involve both heart and pericardium Secondary metastatic growths are much more common and can invade the heart from almost any location in the body Primary growth both benign and malignant types likewise have been reported

### Types

**Primary Growths** About 80 per cent of all primary tumors that invade the heart are benign myomas while the malignant tumors are nearly always sarcomas The benign tumors have been known to arise from the auricles (usually the left) and interauricular septum and the malignant tumors while also showing predilection for the auricles as a site of origin are usually found on the right side The reason is unknown[4] Many of the tumors are pedunculated (see Fig 146) and fill the heart cavity from which they take their origin Some have been found to break off and act as ball valves Others may invade and replace the myocardial structures or may involve the pericardium and compress the heart The conduction system may also be affected and heart block produced (see Case 81)

**Secondary involvement** of the heart by a malignant growth may occur as part of a widespread dissemination or by extension from a

neighboring organ The tumor cells generally reach the heart by way of the blood stream the lymphatic channels playing a much less important role Carcinomatous metastases to the heart are more common than sarcomatous but both occur

**Metastatic growths** may arise from any part of the heart but the right side is more often and more extensively involved than the left The activity of the heart often prevents metastatic growths Later if these processes do gain a foothold their growth is slow, and the heart may compensate for either invasion or compression for a long time When cardiac failure does occur in the absence of a satisfactory etiologic explanation particularly if a malignant process has been previously demonstrated in any organ of the body the possibility of tumor of the heart or pericardium should be kept in mind

## ILLUSTRATIVE CASES

### Cardiac Compression and Failure Following Metastatic Tumor Infiltration—Autopsy

**Case 89** G W a Polish American mechanic of 51 was admitted to the Philadelphia General Hospital on November 9 1938 complaining of cough hoarseness dyspnea and swelling of the feet

History Symptoms appeared four weeks prior to admission when the cough that had been present for 0 years became more severe and was followed by persisting hoarseness Shortness of breath was noted about this time increasing to orthopnea on admission Edema of the feet at night was present four weeks before entry On admission this had advanced to the lower sacral region Past medical family and social histories were negative

Physical examination A thin cyanotic white male B P 94/60 Veins of the neck engorged as well as the veins of the axilla and anterior chest wall Trachea in the midline Chest emphysematous Dulness to percussion over both lung bases Coarse musical inspiratory rales heard over both sides Left cardiac border 14 0 cm to the left of the midsternal line in the sixth interspace Heart sounds weak and distant No murmurs Rhythm regular Liver edge at umbilicus Pitting edema of the lower extremities

Laboratory Data All urine examinations were negative Kahn negative Blood count hemoglobin 78 per cent (Sahli) R B C 4 00 000; W B C 7 00 differential normal Sputum negative for tubercle bacilli

The electrocardiogram (Fig 149B) showed diminution of the amplitude of the QRS complexes

Bronchoscopic examinations showed paralysis of the left vocal cord extrinsic in type There was no evidence of malignancy in the bronchial tree

Roentgen examinations (Fig 149A) (A) the initial study showed a dilated and enlarged heart and fluid in both lung bases (B) three weeks later showed a smaller heart less pulmonary congestion and a large pulmonary conus (C) taken six weeks later showed a diffuse haziness over the left lower hemithorax The left cardiac border was blurred with considerable dense fuzziness about the left hilum Roentgen ray diagnosis Mediastinal adenopathy, most likely malignant displacing the heart

Clinical Diagnosis A Etiologic Metastatic tumor Cardiac compression (?) B Anatomic Cardiac enlargement C Physiologic Congestive failure due to cardiac compression Normal sinus rhythm D Functional Classification Class 4 Therapeutic Classification Class E

Autopsy Ostia and valves unchanged Upper sections of the myocardium were densely infiltrated by tumor The whole thoracic cavity seemed to be crowded by a large tumor mass The layers of the pericardium were involved by the growth and could not be separated The tumor was nodular yellowish white in color and very firm in consistency The large vessels of the mediastinum were patent The microscopic sections showed the invasion of the myocardial structure by the tumor cells There was a primary carcinoma of the lung with metastases to regional lymph nodes and liver as well as the extensions to the pericardium and myocardium

Discussion The increasing dyspnea weakness and cough complained of by the patient centered all attention on the cardiovascular system and




Fig 149 A Roentgen studies 1 November 14 1938 2 December 7 1938 3 January 1939 For explanation see text (Courtesy X Ray Department, Philadelphia General Hospital) B The electrocardiogram Note decreased voltage of the QRS groups and depression of S-T intervals in leads and 3

he was admitted to the hospital as a case of cardiac decompensation Further study however revealed no cause for the failure Finally the aphonia and the nodule observed on the left border of the roentgenogram in the region of the pulmonary artery in the absence of mitral disease

shifted attention from the heart A neoplasm was suspected but from the data available its primary location and exact nature remained obscure

In carcinoma of the lung the possibility of cardiac and pericardial invasion must not be overlooked In a recent study of 1082 cases of malignant disease coming to autopsy Scott and Garvin[337] reported metastasis to the heart and pericardium in 118 or 10 9 per cent Carcinoma of the bronchus and breast made up 48 per cent of these cases In this patient the growth extended from the bronchus and completely encased the heart Invasion of the cardiac structures next took place and practically no myocardium remained (Fig 150) It is remarkable how the residual amount of cardiac structure was capable of sustaining life in this patient for so long a time Interference with diastolic filling finally caused death

Rarely the invasion of the heart by a metastatic tumor growth may involve the conduction system and attract attention to the heart (see Case 81) Although few cases of tumor of the heart are diagnosed correctly prior to autopsy the condition should at least be suspected in the presence of unexplained cardiac failure or heart block significant alterations in the roentgenogram collections of hemorrhagic fluid in the pleural or pericardial cavities or any other cardiac abnormality in a patient recently treated for a malignant process anywhere in the body but particularly in the lung or breast

### Metastatic Growth in Right Auricle and Ventricle from Sarcoma of the Knee—Autopsy

**Case 90** M M a negress of 60 entered the Philadelphia General Hospital complaining of pain in the left leg for five years For the past four years she had lost all motion in the limb and for the past two months had noticed a hard lump on the outer aspect of the left knee the lump had grown rapidly just prior to admission No other symptoms were present

Physical Examination BP 140/90 Rhythm regular rate rapid No murmurs Breath sounds were harsh at the right base and a few scattered rales were present A firm hard mass adherent to the underlying structures was present just above the left knee

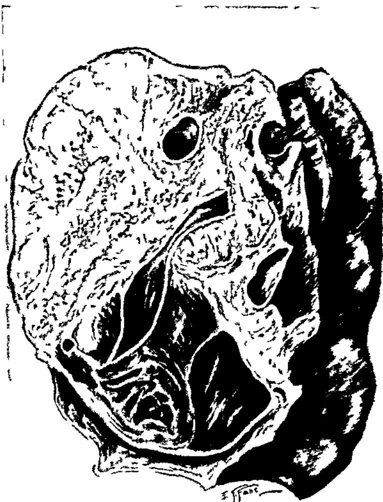
Laboratory Data Wassermann negative Roentgenogram size of the heart shadow was increased with prominence of the pulmonary conus No evidence of metastasis in the lungs Roentgenogram of the leg showed a circular mass in the soft tissues of the lower quarter of the left femur Posterior to the femur in the soft tissues were several dense irregular shadows

The urine on several occasions was negative and the blood count was normal

Autopsy The right auricular appendage contained a growth a long tail of which extended as far as the anterior cusp of the valve The upper and outer portions of the right ventricle were involved in a large mass of growth which extended into the cardiac chamber in the form of polypoid masses The lungs showed metastatic sarcoma

**Discussion** Infiltration of the right auricle and ventricle occurred in this patient and no symptoms were present to direct attention to the heart The only findings were a slight increase in heart size and increase in the prominence of the pulmonary conus in the roentgen picture

The tendency of sarcomatous growths to infiltrate the heart walls and encroach upon the cavity of the heart by the formation of polypoid



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growths is seen in this case (Fig 151) The heart valves were not involved although very rare cases have been reported where tumor cells grow upon the valves simulating subacute bacterial endocarditis

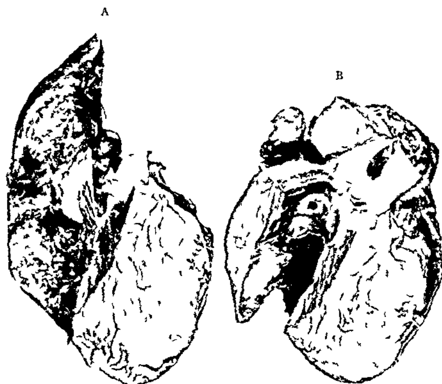



Fig 151 A Tumor growth in right auricular appendage B Invasion of wall of right ventricle A small polypoid growth extended into the right ventricular chamber

## HYPOTENSION

So often patients are seen who complain only of low blood pressure When questioned further the fact is usually brought to light that symptoms began when the diagnosis was made by a physician at the time of a check up for life insurance or an examination for some trivial ailment Complaints of a varied nature are laid at the door of low blood pressure and a great deal of medicine is consumed in attempts to elevate it to a respectable level Many times there is a tendency to lose sight of the fact that low blood pressure is after all not a disease In most instances it is harmless unless mis statements concerning its importance become firmly fixed in the imagination Every physician sees many robust and healthy patients who have systolic pressures of 100 mm of mercury or less

SYMPTOMS AND SIGNS

On the other hand individuals of the asthenic type may be encountered in clinic and private practice who complain of weakness vertigo headache, palpitation and vague digestive tract disturbances They, too may blame their low blood pressure' for every symptom but again we cannot regard the matter as definitely proved, for the low blood pressure may be just as much a part of the picture as the visceroptosis or the small heart There is no reason to believe that the low blood pressure per se accounts for all the complaints and no reason to treat this group as 'essential hypotension

ETIOLOGY

Hypotension may be secondary to a number of important conditions For example we meet it quite often during the course of acute infectious diseases Certain chronic conditions like tuberculosis are also accompanied by arterial hypotension The blood pressure in these instances falls in proportion to the severity of the disease and usually results from the loss of vasomotor tone that accompanies the toxemia The reduction of the blood volume in shock produces a low arterial pressure Myocardial injuries following acute occlusion of the coronary branches are commonly associated with hypotension Various types of endocrine gland disturbances are routinely accompanied by low blood pressure (myxedema Addison s disease) while certain drugs like the nitrite group are capable of producing a sudden lowering of the systemic pressure

**Orthostatic Hypotension** There is an interesting although uncommon condition characterized by sudden fall in the blood pressure when the patient assumes the erect position this may be followed by vertigo faintness and at times collapse I refer to the syndrome orthostatic hypotension Many times patients suffering from this condition will show a decrease in the systolic blood pressure amounting to as much as 50 mm of mercury when they stand There is no compensatory increase in the pulse rate and there is a similar failure of the compensatory mechanism of vasoconstriction which results in an inadequate cerebral blood flow Many normal individuals occasionally experience a sensation of faintness if they suddenly stand erect particularly after a hot bath but this lack of complete circulatory adjustment is not uncommon and should not be diagnosed orthostatic hypotension It differs from orthostatic hypotension in that the circulation makes some attempt to meet the situation The heart rate increases the blood pressure rises at first as the arterioles constrict, but the effort is neither sufficient nor sustained, consequently vertigo and faintness follow the fall in pressure Orthostatic hypotension is caused by a definite impairment in function of the sympathetic nervous system while the faulty circulatory adjustment to the erect posture may occur at some time or another in nearly every normal individual It is mo t often observed *during convalescence from acute infectious diseases*

### TREATMENT

The treatment of hypotension consists of appropriate measures directed toward the cause of the symptoms The patient who has occasional vertigo due to a faulty adjustment to postural changes will be little benefited in the long run by continuous dosing with preparations containing drugs of the vasoconstrictor type Measures should be adopted to prevent the pool ing of the blood on the venous side and to increase the return to the right auricle If we follow Nature s suggestion and place the patient in the hori zontal position when abnormal sensations are experienced improvement is prompt and complete For prophylaxis an elastic abdominal belt and stockings of the same material are beneficial During convalescence from acute infectious disease and other prolonged illnesses graded exercise and massage are of value

**Ephedrine and Benzedrine in Orthostatic Hypotension** Relief may be obtained from drugs that have a vasocontrictor effect The members of this group that have proved mo t efficient are ephedrine and benzedrine Ephedrine may be given in 25 to 40 mg doses by mouth every three or four hours but it must be kept in mind that insomnia is a common unto ward effect that follows large doses of this drug Benzedrine in doses of 20 mg at sufficient intervals during the day to give continuous relief is better for routine use but here too insomnia is often complained of by the patient

Allen and Magee emphasize the importance of an individual program of administration of ephedrine in these cases They show that the drop in the blood pressure when the patient assumes the upright position after use of large doses of these drugs is the same However the level of the blood pressure after the drug has been taken is higher and this accounts for the improvement in the symptoms since the low levels that interfere with cerebral flow are not reached Therefore success in the management of these rare cases lies in keeping the blood pressure at a higher level continuously so that when the fall occurs with the change in position the low level will be above the point where symptoms ordinarily occur

THE TREATMENT OF SECONDARY HYPOTENSION is directed toward the main disease The hypotension of shock for example will respond when meas ures are taken to restore the volume of the circulating blood, while in Addison s disease improvement in the blood pressure accompanies the administration of the hormone from the adrenal cortex

## THE HEART IN INFECTIONS

Infections that cause serious alterations in the cardiovascular system are acute and subacute bacterial endocarditis rheumatism and syphilis The remainder are much less important from a cardiac standpoint rarely pro ducing any serious invasion of the heart Many times the circulatory

disturbances that are observed during the course of acute infectious diseases are brought about by failure of the peripheral vascular apparatus

Quite often the effect of fever on the cardiac structure cannot be distinguished from that of the toxins of micro-organisms The cloudy swelling of the heart muscle observed by the pathologist, as well as the more advanced changes that are characterized by cellular infiltration may interfere with myocardial function When these alterations produced by the infectious process affect the blood supply to the myocardium, more important disturbances often result Toxic substances produced by bacterial growth may also attack the conduction system and heart block may suddenly appear during the course of the infection These changes are usually functional and temporary and disappear as the acute infectious process subsides

As a rule, when the course of the acute infection is a short one the heart is able to perform efficiently and few additional symptoms appear If, however the infection is severe or if it is prolonged signs of congestive failure may complicate the picture at any time (page 194) When these signs are accompanied by a feeble rapid pulse a falling blood pressure increase in the heart size and disturbances in rhythm we may suspect a failing myocardium Tachycardia out of proportion to the height of the fever particularly if accompanied by cyanosis is an early sign that usually directs attention to the circulatory apparatus during the course of an acute infection In pneumonia and other pulmonary diseases cyanosis is not in itself a reliable guide but if at any time it is accompanied by falling blood pressure increasing venous pressure and diminution of the pulmonic second sound a right sided heart failure should be suspected The symptoms on the other hand that suggest peripheral circulatory failure are weakness pallor falling blood pressure decreasing pulse pressure tachycardia and faint heart sounds

Murmurs appearing during the course of acute infections are usually caused by temporary myocardial weakness which permits a relaxation of the mitral ring and mitral regurgitation This functional systolic murmur heard over the region of the cardiac apex many times disappears during convalescence from the infection Rarely are these murmurs associated with actual disease of the heart valve unless the disease is of the rheumatic type

## Treatment

An accurate evaluation of the role of the peripheral circulation and of the heart itself in the production of the symptoms should precede treatment Often this may not be possible when symptoms of cardiac and vasomotor failure suggest the presence of both mechanisms

**Caffeine** administered in the form of caffeine with sodium benzoate in 0 5 Gm (7½ grains) doses is a useful remedy in emergencies It may be given intravenously but the injection should be made very slowly and the drug should be well diluted Direct stimulation of the medullary centers produced by caffeine raises the blood pressure and improves vascular

tone. It also acts directly on the heart muscle, strengthening the systolic contraction, and may increase the cardiac output. A favorable influence may likewise be exerted on the coronary flow. The untoward effects consist of mental excitement and insomnia that are produced in some patients and in the increase in the heart rate. If the tachycardia is marked this may decrease diastolic relaxation and result in diminished cardiac output. The frequency of the administration of the drug should always depend on the effect it produces and the clinical picture of the patient.

**Strychnine** has lost ground in recent years in the treatment of diseases of the cardiovascular system. In the opinion of many the drug is worthless unless used hypodermically in large doses that closely approximate the toxic limit. In single doses of 0.002 Gm. (1/30 grain) to 0.004 Gm. (1/15 grain) it increases nerve irritability, which may in emergencies result in an increase in the tone of the blood vessels.

**Epinephrine hydrochloride** injected intravenously in doses of 0.6 to 1.0 cc. causes increased peripheral vasoconstriction, and this action can usually be relied upon. Intramuscular injections are not as efficient because of the vasoconstriction produced at the site of injection. The action of epinephrine should be a continuous one in peripheral circulatory failure; hence it is best given combined with 500 cc. of physiologic saline or the same volume of 5 per cent glucose by slow intravenous drip (2 cc. per minute). Epinephrine acts directly on the vasomotor nerve terminals to produce its constricting effect and through a similar action on the sympathetics to stimulate the heart. It should be given with great care in cases where the blood volume has been greatly reduced. By increasing the vasoconstriction already produced in these instances, it reduces still further the cardiac output by interfering with the return flow of blood to the heart, and, as Eggleston has pointed out,[90] in this manner aggravates the condition for which the drug was given.

**Ephedrine and Posterior Pituitary.** Both ephedrine and solution of posterior pituitary produce vasoconstriction but are not as certain in their action as epinephrine. Administration of ephedrine sulfate in doses of 0.03 Gm. (3/8 grain) may be attended by excessive nervousness and insomnia, while pituitary extract in repeated doses may be followed by a loss of vasoconstrictor tone and a fall in blood pressure.

**Digitalis and strophanthin** should not be given in cases where the peripheral signs predominate. Even when frank cardiac failure occurs during the course of the infection, digitalis usually has little effect.

**Camphor** and the substances possessing a camphor-like action, such as cardiazol, are of little value. There may be some stimulation of respiration or a slight reflex effect may occur from irritation at the site of injection, but both actions are transient and weak.

In all acute infections preventive measures are more valuable than the remedies above described. Good nursing, proper rest, a well-balanced diet, sufficient fluid intake, and, if cyanosis appears, oxygen by nasal catheter

or tent (page 98) are important in this respect Injections of 50 cc of a 50 per cent glucose solution are helpful When excessive fluid has been lost, the glucose can be given in larger quantities (500 to 1000 cc ) of physiologic saline Blood transfusions under these circumstances are beneficial Great care however should always be used to avoid overloading of the circulation by the injection of too large amounts of fluid When in doubt determination of the venous pressure by the direct method (page 54) may be valuable

### DIPHTHERIA

Both the success in the prevention of this disease and the prompt use of sufficient antitoxin when it does occur make cardiac sequelae rare occurrences today Severe untreated diphtheria however, attacks the myocardium and sudden death in these cases is not unknown The diphtheria toxin causes necrosis of the heart muscle cells and the specialized tissues of the conduction system Arrhythmias produced by invasion of the conduction system furnish clinical evidence of the presence of this destructive process Marked degrees of either auriculo ventricular heart block (page 593) or sinus arrest (page 615) may suddenly appear In addition to the usual clinical signs of heart involvement above described a gallop rhythm may be heard on auscultation furnishing suggestive evidence of prolongation of the conduction time and the toxic state of the myocardium An electrocardiographic study is valuable in revealing the extent of the damage and is generally positive in many more cases of diphtheria than the clinical findings indicate When A V heart block or bundle branch block are present in this disease the prognosis is most serious

Diphtheria toxin may severely damage other structures that contribute to the efficiency of cardiac action and this burden may be added to the myocardial injury For example paralysis of the vagus and splanchnic nerves may augment the tachycardia and add the element of peripheral circulatory failure to the picture

If the patient survives complete healing of the damage produced by the toxin of the diphtheria bacillus in all of these areas is the rule Chronic heart disease does not result from diphtheria Complete heart block if present does not persist and if this conduction defect is found in later life it cannot be viewed as the result of diphtheria in childhood

Early and adequate treatment by antitoxin is the best therapy for the heart in diphtheria In view of the type of damage that has been described a convalescent period of at least three weeks is essential When cardiac damage is extensive in the severe cases small injections of glucose are invaluable The additional measures are the same as are employed when cardiac involvement follows any infection

### SCARLET FEVER

There is a great deal of difference of opinion regarding the production of an endocardial lesion by the streptococcus that has been shown to

stand in an etiologic relationship to scarlet fever The earlier cardiac signs may be associated with the effect of a soluble toxin but close inspection shows that these are all analogous to the change that occur when rheumatic fever follows an attack of acute tonsillitis Even articular manifestations may be associated with scarlatinal infection although these are milder are associated with less effusion and tend to involve smaller joints The valvular involvement that is described as scarlatinal is identical with rheumatic endocarditis Similar electrocardiographic alterations are produced Consequently the bulk of present day evidence points to the fact that scarlet fever is merely the activating agent The patient quite often has a rheumatic constitution and a positive family history

I observed a similar response following erysipelas involving the face and scalp in a child of seven Two weeks after the initial infection subsided leaving the boy as bald as an onion a typical attack of rheumatic fever with joint manifestations developed A mitral lesion was discovered a month later During the course of the intervening years the development of mitral stenosis has been followed The last examination 20 years after the attack of erysipelas showed mitral stenosis and mitral insufficiency with considerable cardiac enlargement

### INFLUENZA

Although many symptoms of a cardiovascular nature are frequently observed following influenza we cannot say that this disease causes any permanent damage A cardiac examination at the height of the disease will reveal the usual changes associated with any other febrile disorder with the exception perhaps of the relative bradycardia During the course of influenza other arrhythmias such as premature contractions and marked sinus irregularities are not infrequent The pathology in this disease is obscure However if structural alterations do occur as some observers claim they completely disappear following convalescence

The postinfluenzal symptoms are usually shortness of breath precordial distress palpitation weakness and exhaustion All appear on slight exertion and may be regarded in most instances as manifestations of neurocirculatory asthenia (page 410)

### PNEUMONIA

The pneumococcus infection does not in itself cause serious heart involvement unless the endocardium is attacked (page 185) or suppurative pericarditis develops following extension of the process from the lung (page 171) Pneumonia may prove to be a most serious complication in the presence of advanced heart disease The cardiac failure occurring late in the course of this disease usually shows a poor response to the usual measures Routine digitalization of all pneumonia cases is not recommended However the use of the drug in the presence of auricular flutter auricular fibrillation or congestive failure is indicat

sugar salt and oxygen are the most useful remedies in the treatment of the circulatory collapse in pneumonia

### TUBERCULOSIS

The effect of tuberculosis on the heart may be considered from many angles The tubercle bacillus itself only rarely attacks the heart When it does the pericardium and myocardium are invaded by direct extension from a neighboring focus or as a part of a general miliary involvement In the course of the latter process the endocardium may be seen to contain small tubercles of the miliary type at autopsy They are never important clinically However the pericardial process may heal in some cases with the subsequent deposition of calcium and by its constriction produce grave interference with diastolic filling (page 182)

In some cases an active tuberculous infection may produce alterations in either rate or rhythm of the heart that will attract attention of the patient toward the cardiac mechanism (case 64) Neurocirculatory asthenia not uncommonly accompanies tuberculosis

Alterations in the lungs during the course of the disease may in some cases place a strain on the right side of the heart and the symptom of chronic cor pulmonale may be observed (page 426) In other instances a sudden pneumothorax may produce pain suggestive of coronary occlusion If the mediastinal structures are displaced in advanced phthisis by adhesions a variety of confusing cardiac signs appear

Tuberculosis of the lungs is not evidence against the diagnosis of organic heart disease However because of the bed rest usually prescribed for the tuberculous patient congestive failure seldom appears Pulmonary tuberculosis often complicates congenital stenosis of the pulmonary valve (page 346) as a result of inadequate circulation in the lungs Likewise in engorgement of the vessels of the lung that is seen in mitral stenosis, pulmonary tuberculosis is rare but has been observed

The treatment of active tuberculosis of the pericardium and the heart has been discussed elsewhere (page 175)

### FOCAL INFECTION

The concept that infection in the teeth tonsil sinuses gallbladder cervix or gastro-intestinal tract is responsible for a variety of diseases has gained headway in recent years Although we possess no entirely satisfactory criteria for determining the presence or absence of infection in many of these localities the teeth and tonsils seem to bear the brunt of the attack of the focal infection enthusiasts Consequently many unnecessary operations have been performed that are not without danger in patients who have heart disease Bacteria gain entrance into the blood stream at times following these procedures and in some cases a fatal endocarditis results (page 197)

All the diseases of unknown etiology that affect the heart have at one time or another been blamed on the tonsils Reimann and Havens[310] point

out that one third of all surgical operations performed in a group of nearly 40 000 cases were tonsillectomies The extraction of diseased tonsils prevents the recurrence of quinsy and acute tonsillitis and may decrease the frequency of attacks of acute rheumatic fever However we must remember that this procedure does not offer to all patients a guarantee against the occurrence of rheumatic infection and subsequent heart disease Many studies of groups of tonsillectomized children that have been made by Kaiser[1 8] and others[3] reveal no great decrease in the incidence of rheumatic heart disease following operation when compared to control groups In view of the number of tonsillectomies performed today if this were the solution of the problem of rheumatic infection the incidence of this disease by this time ought to show a decided decrease Such is not the case On the other hand tonsillectomy itself may be followed by a number of serious complications By no means the least important of these is a violent flare up of a smoldering rheumatic state

It is unfortunate that the wide acceptance of the attractive theory of focal infection has elevated it in the minds of many to the position of a proved fact I believe that the treatment of heart disease by the eradication of these foci should always be governed by the circumstances in each case For example pain tenderness swelling and lymph node involvement speak for the presence of infection in a tooth and its removal should be considered Roentgen films alone should not decide the issue for they are often unreliable in these cases Similarly only tonsils that show a persistent redness accompanied by injection of the anterior pillars painful swallowing enlarged regional lymph nodes and systemic evidence of infection should be considered diseased and their removal recommended I do not believe that either the size of the tonsils or the cheesy material expressed from their crypts should be accepted as adequate evidence for a mandatory tonsillectomy As a matter of fact the debris that we so often demonstrate in the tonsillar crypts is harmless When treatment is begun for the patient s condition as a whole the lymphoid tissue in the pharynx and throat that previously suggested the presence of infection may take on a quite different appearance Needless to say surgical treatment of all foci of infection should be delayed until a complete survey of the patient has been made Surgical attacks on accessible foci in cardiac cases should never be recommended unless the evidence in each case leaves little doubt as to the presence of infection Prophylactic tonsillectomies are not justified The same rule should apply with respect to the teeth It still remains to be proved that any primary disease of the heart is the direct result of infection in the teeth

# 15

# THE HEART IN PREGNANCY

In the management of the cardiac patient during pregnancy many situations are encountered that demand a thorough knowledge of the diagnosis and prognosis of heart disease Numerous questions are likewise asked by patients contemplating marriage that call for an appraisal of the circulatory status and an estimation of the probable effect of the additional burden of pregnancy Certain rules of practice may be adopted in applying our knowledge of heart disease to these special situations These may be better understood if the fundamental principles upon which they are based are first briefly discussed

## THE PROBLEM

During pregnancy the heart has more work to do This is not the result of any single factor but is brought about by the many physiologic alterations that take place as gestation advances To begin with the gain in weight accompanying pregnancy places an extra demand on the circulation Moreover since this gain is also a local one the postural alterations produced may have a tendency in some individuals to add an extra burden during the course of pregnancy Too much significance however should not be placed on this single factor of increased body weight as a cause of heart strain for Jensen has cited four cases from literature where improvement in the circulation took place at the time of fetal death and not following delivery [1]

Aside from the patient s increase in weight the continued advance in uterine size with its increasing vascularity is an additional factor that adds to the work of the heart From this standpoint as Burwell has pointed out [47] pregnancy increases the cardiac burden by the development of wide communicating channels in the uterine wall in the manner of an arteriovenous fistula

Successive pregnancies probably have no permanent effect on the vascular bed itself There is nothing to prove that a "toxin is produced to activate changes in this area or to produce even a temporary vasoconstrictor effect that may increase the cardiac load

With elevation of the basal metabolic rate extra work is placed on the heart in fact the operation of total thyroid ablation was planned to decrease the metabolism and improve the circulation by decreasing the cardiac load In pregnancy the thyroid gland enlarges and it is natural to suppose that an increase in the work of the heart takes place and accompanies the increase that is observed in the basal metabolic rate

Recent studies confirm the fact that the circulating blood volume is increased during pregnancy Although there is some difference in the figures obtained by the use of the various gas and dye methods the increase in some cases has been found to be as high as 40 per cent Consequently, again we have proof of an increase in the cardiac load

Clinical and laboratory studies indicate that the normal heart adequately meets these demands placed upon it during pregnancy A slight increase in the blood pressure particularly the diastolic balances the increased peripheral resistance the pulse rate quickens, and the minute volume increases while the circulation time remains unchanged

## EXAMINATION OF THE HEART

If we apply the ordinary methods of examination to the heart in pregnancy to detect what changes if any take place the result is interesting and will lead us into a great deal of speculation

**Change in Size of Heart** In the first place the medical pendulum seems to be unwilling to come to rest in regard to the question of the presence or absence of cardiac hypertrophy However much depends on our conception of the word hypertrophy The heart does increase in size but this is entirely within the limits that would be expected when viewed in the light of the increase in body weight The heart under the fluoroscope appears larger in fact the actual measurements are often increased It must be realized however that as pregnancy continues the heart is elevated and perhaps rotated by the high position of the diaphragm under which circumstance accurate determination of the cardiac size becomes difficult (Fig 152) The cardiac shape may in some patients be considerably altered by these changes and present the appearance of mitralization (page 37)



FIG 152 Alteration in cardiac position as pregnancy advances Dotted line indicates heart's position at third month The solid line shows position two weeks prior to term

**Change in Position of Heart** This shift in the position of the heart may be reflected in the change in the electrical axis of the electrocardiogram which must not be viewed as evidence in favor of hypertrophy In pregnancy there is a tendency for an inversion of the P wave and T wave to occur in the third lead and Pardee has called attention to a deep Q-wave in the same lead that may accompany a normal pregnancy (Fig 153) All these changes in the electrocardiogram may be explained on the basis of the change in cardiac position

**Appearance of Murmurs** The most common finding in the obstetrical department that causes the patient to be referred for cardiac study is the presence of a murmur Since this physical sign continues to be so intimately linked with heart disease in the minds of most physicians furthe

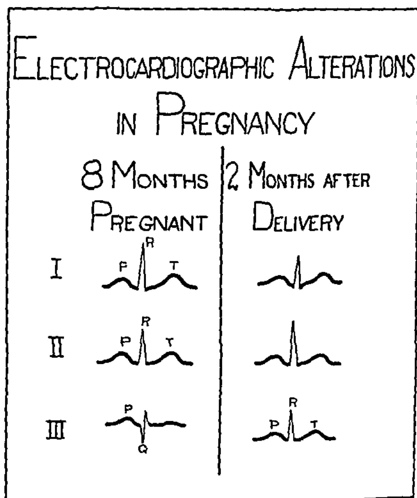



FIG 153 The electrocardiogram in pregnancy

special study of these patients is requested During pregnancy a systolic murmur over the pulmonary area should cause no alarm It is produced in many instances by the shift in cardiac position that is due to the steady elevation of the diaphragm It is usually loude t in recumbency and may almost disappear when the patient is in the erect position This pulmonary systolic murmur appears after the fourth month of pregnancy At times these functional systolic murmurs may be heard over the whole pre

cordium but they are usually loudest in the second interspace to the left of the sternum Functional diastolic murmurs have been described but I have not heard them The pulmonic second sound may be accentuated during pregnancy because of the change in cardiac position that serves to advance the valve nearer the chest wall where its sound is more clearly heard Before a diagnosis of heart disease is made during pregnancy these physiologic alterations should be kept in mind Examination of the heart during pregnancy is not easy since enlargement of the breasts makes percussion and auscultation more difficult

**Other Diagnostic Signs and Symptoms** To confuse the picture further other reliable diagnostic signs and symptoms are of less value during pregnancy for example edema and dyspnea

It is not at all uncommon for edema of the feet to occur in normal women toward the end of pregnancy because of pressure on the pelvic veins and alterations in the capillary bed of the extremities

Likewise when dyspnea is complained of allowance must be made for the high position of the diaphragm that crowds the lungs and for the other anatomic and physiologic alterations just described In the cardiac examination during pregnancy reliance must be placed on diastolic murmurs either apical (mitral stenosis) or basal (aortic regurgitation) harsh apical systolic murmurs in patients with positive rheumatic histories engorgement of the neck veins enlargement and tenderness of the liver thrills unmistakable displacement of the apex beat or widening of the heart at the base serious arrhythmias like auricular fibrillation auricular flutter pulsus alternans or heart block friction sounds and the signs of chronic hypertension or nephritis

**Etiology** The majority (80 per cent of our series) of patients of child bearing age who have heart disease suffer from the rheumatic type with mitral stenosis Active rheumatic disease however is rare during pregnancy Congenital defects are present in a very small percentage of the cases whereas hypertension nephritis syphilis and arteriosclerosis can be grouped together as infrequent causes of cardiac complications (Table IX)

Table IX

TYPES OF HEART DISEASE

| | Number | Percentage |
|---|---|---|
| Rheumatic | 32 | 80 |
| *Congenital* | 2 | 5 |
| Hypertensive | 2 | 5 |
| Miscellaneous | 4 | 10 |

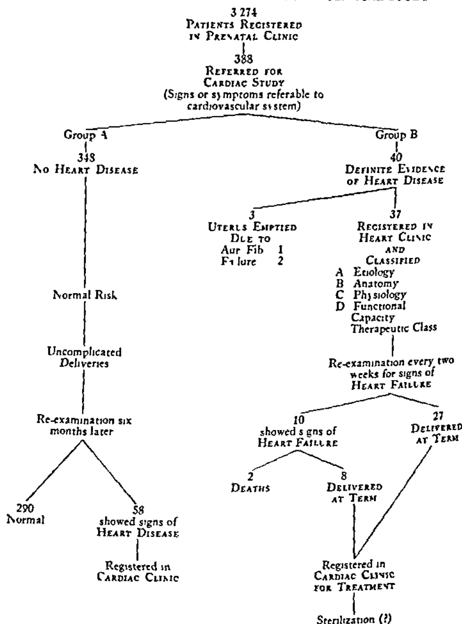
## CLASSIFICATION

For purpose of diagnosis and treatment a working classification of the patients presenting signs or symptoms of heart disease during pregnancy is essential Prenatal patients referred to the Cardiac Clinic of the Woman s

College Hospital after the first examination are divided into Groups A and B (Table X)

TABLE X

HEART DISEASE DURING PREGNANCY—A CLINICAL STUDY




**Group A** Patients who have systolic murmurs questionable degrees of edema, palpitation or dyspnea together with a negative rheumatic history

and no abnormal alterations in cardiac size or contour are placed in Group A The presence of heart disease cannot be proved and these patients (we assure the obstetricians) are excellent risks We tell the patients that they have normal hearts as far as we are able to estimate during pregnancy They are urged however to return for one examination following delivery

**In Group B** are placed all the patients who have cardiac enlargement diastolic murmurs and any of the signs of organic heart disease above mentioned We separate the patients belonging to this group and focus our attention on them for they are the ones most likely to develop cardiac complications as pregnancy advances We do not include in Group B any of the cases of mitral regurgitation and for purposes of this study we have allowed a large number of these patients to remain in Group A Group B is subdivided according to the classification of the American Heart Association (page 59) We consider this system most useful inasmuch as the obstetrical risk is in large part governed by the presence or absence of manifestations of congestive failure Likewise placing the patient in one or the other of these groups after the study is completed is a matter of great convenience It helps to establish proper treatment at an early date but it does not necessarily mean that the patient will remain permanently in the group nor does it indicate that the management will be group management and not adapted to suit the needs of the individual patient Seventy five per cent of our Group B patients belong in Class I 20 per cent in Class II 3 per cent in Class III and 2 per cent in Class IV

## MANAGEMENT

Patients in Group A do not of course require any special care as far as the heart is concerned

In Group B the patients in Class I and many in Class II if carefully watched can usually be counted upon to come through pregnancy and delivery without complications They report to the Cardiac Clinic once a month until the sixth month and are then seen at intervals of two weeks until they enter the hospital for delivery

**Rest** should be prescribed for Group B patients to reduce the demand made upon the heart to a degree consistent with the estimated reserve In the absence of any signs of congestive heart failure however an excessive amount of rest is detrimental to the patient Exercise of mild degree in the open air assists venous return to the heart and is decidedly beneficial

**General Measures** Group B patients are advised to avoid upper respiratory infections as much as possible As pregnancy progresses the household duties are lightened in each case to allow the patient to keep within the limits of her cardiac reserve This is estimated at each follow up visit when the progress notes are recorded and the examination of the patient is completed

**The laboratory procedures** indicated in cases where cardiac disease

is complicated by pregnancy include urinalysis Wassermann electrocardiogram and orthodiagram When these are obtained and evaluated the patient can be properly classified and her program outlined

**Follow up visits** give opportunity to check for errors in classification caused either by a mistake in interpretation of the findings or by a change in the patient s status as pregnancy advances On these occasions the blood pressure the respiratory rate pulse rate, and body weight should be recorded The feet should be inspected for signs of edema and the lung bases carefully examined If rales persist in the lung bases bed rest for two or three days followed by re examination should be the routine prescription The electrocardiograms usually do not have to be repeated unless arrhythmias are present that elude clinical detection

## Delivery

All Group B patients are admitted to the hospital two weeks before term for rest and additional observation Pregnancy is allowed to terminate normally Most of Class I and many of Class II patients deliver spontaneously with little trouble although the second stage of labor is often shortened, when indicated by the application of forceps Ether anesthesia can be used safely for the whole group The longer convalescent period allowed in the hospital usually means that the majority of these patients are discharged in excellent condition Unless an earlier date is indicated for some special reason they return in six weeks to the Cardiac Clinic for the first follow up study

Patients in Classes III and IV require close attention The prognosis of Class III patients is guarded while those in Class IV usually have a poor prognosis The complication that causes most concern to internist and obstetrician who work together in these cases in Classes III and IV is congestive failure It is responsible for 74 per cent of fatalities according to the recent report of Carr and Hamilton[5] Pneumonia pulmonary infarction sepsis and embolism are more frequent complications in these patients because of the influence of the congestive failure

**When congestive failure** appears it is usually during pregnancy and rarely during labor when the strain is supposed to be the greatest The reason may be better understood when we consider the fact that the patient is in labor a short time compared to the long period of pregnancy Nevertheless the influence of a poorly conducted labor in precipitating congestive failure must never be underestimated An upper respiratory or other infection may often play a large part in imposing the *additional* burden that upsets the patient The earlier in pregnancy congestive failure appears, the poorer will be the prognosis More frequent prenatal visits of the cardiac patient to her physician are mainly for the purpose of detecting the signs of failure as early as possible If during pregnancy a patient is seen presenting signs of advanced decompensation some one has usually fallen down on the job

The early recognition of congestive failure in pregnancy is not easy

but is possible if careful examinations are carried out. The symptoms of dyspnea and palpitation together with the signs of tachycardia and edema must all be evaluated in the light of the physical state. On the other hand congested neck veins orthopnea gallop rhythm pulmonary edema pulsus alternans are all advanced signs of failure. Sudden paroxysmal dyspnea is rare as a complication of rheumatic heart disease but can occur in the presence of pulmonary overloading produced by a strong overactive right ventricle. The initial attack occurred during labor in one patient of our series who suffered from hypertensive cardiovascular disease. Although this patient was successfully delivered death occurred during another seizure two months after discharge.

At times in doubtful cases estimation of the venous pressure is a very great help in the early diagnosis of congestive failure (page 54). However congestion of the lungs should be suspected when persisting rales are heard at the bases at any routine visit during pregnancy. Rales should always be regarded as a warning of approaching danger and bed rest and digitalis prescribed. The speed of digitalization is always determined by the condition of the patient. When improvement takes place the drug should be continued in maintenance dosage until the time of delivery. Pregnancy offers no contraindication to the use of the usual diuretics including the mercurial group.

The extent of the bed rest will depend on the response shown to the treatment. If improvement is rapid the patient may be allowed to be about again at an early date. Some patients however may have to remain at nearly total bed rest until delivered. At term the patient who can undertake but little exertion without great discomfort is in no condition to stand labor and should not be allowed to deliver spontaneously.

If the patient when first seen is in severe congestive failure medical treatment should be given a thorough trial since the first aim of the physician should always be to restore cardiac balance. Attention is then directed toward the termination of the pregnancy if there has been no adequate response to the measures employed. However let me hasten to add that medical treatment seldom fails to restore enough circulatory balance to make subsequent intervention a much less dangerous measure. Haste in these situations only increases the maternal mortality rate.

**If auricular fibrillation** is present as Mackenzie originally stated [1] the prognosis should be considered more serious. However the management remains the same. The attempt should be made to control the ventricular rate by full doses of digitalis and if the usual satisfactory result *is obtained* operative interference can be delayed until a rest period improves the risk.

**The additional measures** to be considered in the treatment of the decompensated patient during pregnancy differ very little from those already outlined. Dietary regulation (page 548) has for its guiding principle the administration of suitable amounts of glucose. In cases where the heart failure is attended by considerable cyanosis oxygen if available will be of

great benefit since it also increases the chances of survival of the baby (page 98) A flow of four liters per minute using the nasal-catheter apparatus is satisfactory in emergencies Higher concentrations of oxygen should be used with great care in these patients since nearly all of them suffer from advanced mitral stenosis The danger of a higher percentage lies in producing bronchial irritation cough and pulmonary edema

**Termination of Pregnancy** Many facts are to be carefully considered before choosing the method to be used in terminating pregnancy Each case must be decided on its own merits for in this matter no set of rules can be laid down and dogmatically followed A great deal depends upon the time the patient is first seen If very early in pregnancy and congestive failure is already present the outlook is serious and therapeutic abortion is the method of choice after careful preparation of the patient In further advanced borderline cases the history of previous pregnancies may serve as a guide to the procedure However, if in doubt concerning the method of choice it is always best to do nothing, since patients with congestive failure who are subjected to the additional shock of ill timed interference invariably do poorly Therapeutic abortion should never be performed until congestive manifestations clear If labor begins spontaneously to further complicate the situation morphine should be given in an attempt to postpone delivery until circulatory balance has been restored

**Cesarean Section** In Class III and Class IV patients the operation of cesarian section has much to recommend it It can be carried out under spinal or local anesthesia depending on the preference of the surgeon and in well prepared patients can be accomplished in a very short time with but little additional strain on the circulation Of course the dangers of hemorrhage shock anesthesia and embolism are added to a poor risk but I believe that these are more than overbalanced by the strain of a long labor

**Sterilization** can be carried out at the time of operation if cesarean section is the method of choice Sterilization has much to recommend it and should always be done if the heart condition is too severe to stand the strain of a normal delivery (Class III and IV patients) and when the patient with heart disease already has more children at home than her fast diminishing cardiac reserve will permit her to care for efficiently Here again the factors present in each case will guide the selection of the proper procedure

**Anesthetic** There is little to fear from the anesthetic if it is wisely chosen and properly administered Ether by the open drop method is safe in the majority of cases Chloroform adds a risk that is not negligible Nitrous oxide and oxygen mixture may be used if skillfully given while ethylene has furnished good results in clinics that are properly equipped for its use As a general rule anesthetics administered by mouth or injection are not as satisfactory as the ones administered by inhalation Once injected the action is harder to control, and in some cases postoperative complications occur No matter how trivial the operative procedure or how good the risk appears to be cardiac cases at all times require careful

handling Consequently psychic shocks worry insomnia are all indications to postpone operations until the patient is in better condition Neglect of this last principle may occasionally be followed by disaster

The use of pituitrin to speed labor in cardiac patients is to be condemned for in the conduct of labor in the presence of advanced heart disease speed is not as essential as the conservation of the patient s energy Observation of the blood pressure respiratory rate and heart rate should be made frequently during labor The neck veins and the patient s color should be watched closely Increase in the heart rate congestion in the neck veins and rales in the chest are danger signals Many cardiac patients are unable to stand the recumbent position and should be delivered with the back of the table elevated to about 30 degrees The Trendelenberg position should never be used

Usually if cardiac failure is avoided by the combined efforts of cardiologist and obstetrician during pregnancy it does not appear during labor or the puerperium If it does appear after delivery some other precipitating cause must be suspected and searched for Sepsis infarction phlebitis and mastitis are a few of the more common causes to be kept in mind

### Puerperium

The patient must not be neglected during the puerperium since following the marked fall in the intra abdominal pressure and the consequent shift in the cardiac axis circulatory accidents may be occasionally encountered Sand bags on the abdomen are recommended by some obstetricians to maintain pressure and lessen the shock of sudden circulatory readjustments

## SHOULD PREGNANCY BE CONTEMPLATED BY THE CARDIAC PATIENT?

This question is difficult to answer offhand but may be satisfactorily worked out for each individual case when all the facts are carefully set down The type of heart disease present and its severity should first be considered If advanced rheumatic heart disease is discovered and the cardiac reserve is low or if the patient has already survived one or more attacks of congestive failure the decision must be in the negative In established but less advanced cases the decision may be more difficult If the patient has had rheumatic heart disease for some time and has been under the constant supervision of the family physician no one is better suited to give this opinion Previous entries on the office card give valuable clues to the degree of progress of the patient s cardiac lesion and are most useful If the decision is still difficult it is best to make a mistake on the safe side than to be too optimistic Generally it is satisfactory to place the facts squarely before the patient and her husband and outline the risks that must be assumed The station in life and the economic condition of the

family in question are points to be weighed in the balance In other word will the patient be in a position to take good care of herself during the pregnancy Unfortunately for each patient who seeks this advice before marriage the physician will have many who come in for advice when pregnancy is already present

Opinions governing subsequent pregnancies cannot be entirely based upon observations made during the initial one The cardiac lesion since it is usually of the rheumatic type may be progressing in which event the reserve diminishes Many times it is difficult to say whether this is the effect of the first pregnancy on the heart or whether it is the result of an advancing rheumatic infection If some years have gone by since the first pregnancy the age of the patient must be given due consideration, since older patients with the same degree of damage do not do as well as the younger ones Finally it may be stated as a general rule that if the heart lesion is trivial the patient may have two or at the most three children If on the other hand the lesion shows signs of progressing and particularly if the patient is over 30 she should realize that the second pregnancy may not be as smooth as the initial one and that the risk assumed is far greater She will then usually be content with one child I must admit that I have seen many instances where a second pregnancy was successfully weathered in spite of advanced rheumatic heart disease when the patient had been warned against it

Gilchrist[11] studied 109 cases of fatal cardiac rheumatism in order to discover the effect of repeated pregnancies on the course of the disease A comparison was made with males nulliparae and parous women regarding the average age of death mode of dying duration of the cardiac disease and the rate of progression to fatal termination No significant difference was found in the duration of the disease in nulliparous and parous women Auricular fibrillation Gilchrist found is not necessarily an indication of an additional burden placed on the heart during the child bearing period Its incidence he believes is largely determined by the length of survival from the time of the first involvement of the heart Women dying from congestive heart failure (the mode of death in 92 per cent of the whole group) had families averaging 4.5 children each a fact that supports the contention that the strain of frequent child bearing brings earlier death Gilchrist concludes that one or perhaps two pregnancies have little effect on the course of the disease in the majority of the cases but repeated pregnancies will definitely shorten the life span of women suffering from rheumatic heart disease since they increase the incidence of congestive cardiac failure

### SUMMARY

### The Heart in Pregnancy

#### I The Risk

1 All patients in Group A (Table X) do well If carefully managed those in Group B (Classes I and II) give little trouble

2 In patients who have established mitral lesions (usually stenosis) with little or no cardiac enlargement the risk is slight

3 As a rule the risk is directly proportional to the size of the heart There fore routine fluoroscopic examinations should be made during pregnancy on all patients who have established cardiac lesions

4 The most important single factor in the determination of the risk is the cardiac reserve This can be estimated by inquiry concerning patients ability to perform ordinary household duties without discomfort Valve lesions *per se* have little influence on the risk

5 Danger signals Signs of failure appearing at any examination auricular fibrillation or great increase in cardiac size (Apex beat or left border in axilla) and a previous history of congestive cardiac failure

6 Good prenatal care and co operation of patient will greatly improve the risk in many cases and permit delivery of a normal child at term

II RULES FOR MANAGEMENT

1 If cardiac lesion slight one or two pregnancies permitted If cardiac lesion moderate one pregnancy permitted Pregnancy should be for bidden in cases showing

(a) Previous history of heart failure

(b) In presence of an advanced lesion with auricular fibrillation

2 When patient who presents contraindications to pregnancy is seen during the first three months empty the uterus If seen later treat congestive failure (bed rest and other measures) then do cesarean section OPERATIVE INTERFERENCE IS CONTRAINDICATED IN THE PRESENCE OF CONGESTIVE FAILURE!

3 The patient should be examined frequently during the later months of pregnancy IT IS EASIER TO PREVENT CONGESTIVE FAILURE THAN IT IS TO TREAT IT!

4 For delivery or operative interference inhalation type of anesthetic is preferable ether ethylene alone or combined with local

5 When pregnancy is complicated by a cardiac lesion the best results are obtained when pregnancy is allowed to follow a normal course The second stage of labor may be shortened by forceps

6 Induction may be a more serious trial than normal labor at term Acute left ventricular failure followed attempted induction in one case in our series

# 16

# ALLERGY AND THE HEART

By RICHARD A KERN, MD*

There is as yet no proof that the heart itself can be the site of an allergic reaction That is to say no one has demonstrated in the heart after exposure to an allergen the occurrence of edema or of smooth muscle spasm the basic phenomena of the allergic response There is, however good clinical evidence that an allergic reaction may be the trigger mechanism which may precipitate certain symptoms referable to the heart notably some of the cases of paroxysmal auricular tachycardia A similar relationship has been suggested between an allergic reaction and other cardiac diseases but the evidence is far from convincing Of major importance from the viewpoint of clinical medicine is the very serious role which allergic disease especially of the respiratory tract so often plays in patients who are suffering from some form of cardiac disorder

## CARDIAC CONDITIONS PROVOKED BY AN ALLERGIC MECHANISM

It is an axiom that the purely allergic reaction is wholly reversible An urticarial wheal disappears without leaving behind it any trace of structural change The nasal mucosa which for weeks has been the site of marked edema during the hay fever season reverts to normal within a few hours of the removal of the offending pollen from the inspired air The smooth muscle of gut or bronchial tree even after many episodes of allergic spasm shows at most a very trifling and usually no hypertrophy

If this reversibility of an allergic reaction be as true of a possible cardiac allergy as it is of allergic manifestations in other organs and tissues then in the intervals between allergic attacks the heart should be normal according to every criterion of examination Furthermore it should be possible to elicit the allergic response on adequate exposure to that to which the person is sensitive and he should remain free of symptoms so long as such exposure is avoided

These conditions are rarely fulfilled in patients known to be allergic and who have symptoms of cardiac disease As a rule such patients give some evidence of structural or functional cardiac abnormality throughout the interval between allergic episodes At this point the question might be raised Will not a permanent structural change result at times from frequent repetition or prolonged duration of an allergic response as for

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example nasal mucous polyps in perennial allergic rhinitis? True but before that final stage of polyposis is reached there has been a long period without permanent structural change simply an edema which promptly subsides when there is no longer any exposure to the allergen which provoked it Only rarely, even during the beginnings of symptoms does one encounter in the intervals between allegedly allergic cardiac episodes a heart that is altogether normal in structure and function As a rule one gets the impression that the allergic reaction is an added factor which provokes symptoms in a heart that has already been damaged by some other disease process

Even when the heart appears to be normal between attacks one is not justified in concluding when cardiac symptoms are produced on exposure to a substance to which the patient is sensitive that the heart itself is the site of the allergic reaction The reaction might have occurred elsewhere and might have affected the heart indirectly by way of nerve pathways This will be discussed further under paroxysmal auricular tachycardia

This discussion may seem unduly theoretical for a clinical text but it has this fundamental significance If the cardiac condition were due only to allergy then its management would call for treatment primarily and perhaps solely along allergic lines But if as is the rule one is dealing not only with allergy but with a heart that is the site of some additional pathologic process then allergic therapy is only an incident in the management of the underlying cardiac disease

## PAROXYSMAL AURICULAR TACHYCARDIA

This is perhaps the outstanding cardiac disorder in which an allergic mechanism may play a part The systematic description of paroxysmal auricular tachycardia its etiology diagnosis and treatment will be found in Chapter 12 (page 382) the present discussion is limited to the allergic aspects

In a series of 28 cases of paroxysmal tachycardia seen by me eight gave sufficient evidence of food allergy to warrant the conclusion that the allergy played a major role in the production of the arrhythmia Criep (personal communication) has had a similar experience L P Gay[11] has also encountered cases of this type The following patient reported by me in 1932[18] well illustrates the salient points

## ILLUSTRATIVE CASES

**Case 91** Miss E S a nurse aged 40 years was seen by me on December 9 1931 in the midst of an attack of tachycardia She had been subject to such attacks as long as she can remember at least since the age of five They have been characterized by sudden onset of rapid heart action usually in relation to indigestion The indigestion was usually caused by eating nuts which always upset her The attacks stop in a few minutes as suddenly as they start usually after the use of one or another maneuver upon which experience has taught her to rely The most effective of these has been to st n[illegible]

leaning forward with knees straight and finger tips touching the floor The present attack had begun the preceding evening while attending a dinner dance at which she had eaten freely of nuts Being loath to try the above described stunt on the dance floor she waited until she got home only to find that it would not work When I saw her at one o clock the next morning the attack had lasted four hours the pulse being 180 I prescribed capsules of quinidine sulfate 0 Gm one to be taken every four hours The first dose produced some nausea and itching The second was followed by similar but more severe symptoms and a crop of hives The heart rate dropped suddenly to normal within an hour after the second dose

When seen on January 1 193 73 days after the attack she was normal in every respect There were no discoverable abnormalities in heart or circulation and her circulatory response to exercise was excellent She had always been much given to athletics *including swimming golf and riding and has never experienced any cardiac limitations* in these activities An electrocardiogram on this date showed a normal tracing

The following significant facts and data were subsequently established all her life she had been subject to hives Her mother had typical and severe migraine Skin tests showed her to be skin sensitive to some and not to others of the foods which could provoke attacks of tachycardia Avoidance of these foods was followed by comparative freedom from attacks for a number of years

On January 5 1940 after a banquet at which she ate not wisely but too well she developed another severe attack of tachycardia The second dose of 0 1 Gm of quinidine sulfate stopped the attack which had then lasted nearly ten hours *The smaller dose of* quinidine produced nausea and some itching but no hives

**Discussion** This case illustrates several points which are highly important in clinical problems involving the proof of an allergic factor in the production of a specific disease picture

1 The Patient Is Allergic For years she has experienced attacks of an obviously allergic condition hives She is probably of allergic heredity her mother having had typical migraine, which is commonly of allergic etiology An allergic individual is one who is born with the characteristic *of becoming sensitized to things in his environment more easily than* do normal persons This characteristic of easy sensitizability may manifest itself at any time during the patient s whole life time and in a wide range of tissues and organs

2 In an Allergic Patient Therefore Allergy Must Be Routinely Suspected as a Possible Etiologic Agent in Many Illnesses for example paroxysmal auricular tachycardia in this patient

3 The Clinical Test Gives the Necessary Proof of an Allergic Etiology When she avoids those things to which she is sensitive, she is symptom free When she eats the offending foods she develops attacks of tachycardia

4 The Patient Is Sensitive to Quinidine Drug idiosyncrasy occurs far more frequently among those who are obviously allergic The drugs to which patients are most commonly sensitive are aspirin quinine and the iodides Those sensitive to quinine are usually sensitive to its derivative quinidine If one third of cases with paroxysmal auricular tachycardia are associated with allergy then the chances of encountering such clinical idiosyncrasy are not to be overlooked All prospective candidates for quinidine therapy should be questioned as to a possible quinine idiosyncrasy In case of such idiosyncrasy great caution and a sharply reduced dosage

are called for In this same connection attention is called to the fact that acetyl beta methyl-choline and related substances which have proved effective in arresting paroxysms of tachycardia may at times precipitate an attack of asthma in those inclined to that disease They should therefore be used with caution in all allergics and preferably not at all in those who are obviously asthmatic (see Case 71)

5 AFTER COUNTLESS PAROXYSMS OF TACHYCARDIA DURING MORE THAN 40 YEARS HER HEART CONTINUES TO BE NORMAL The manifestations of allergy are primarily functional and reversible Except under extraneous unfavorable circumstances (e g infection in allergy of the respiratory tract) they do not lead to organic change At this point the question could be asked might this not be an instance in which the allergic reaction actually takes place in the heart itself? Perhaps But the facts do not warrant the conclusion, since the same effect might have resulted through a reflex pathway from an allergic reaction elsewhere in the body The following case reported by me in 1926[196] illustrates the point

**Case 92** A woman of 35 years of age ith no previous h story of card ac d sease and no evidence of subsequent development of such d sease in the e ght years thereafter was subjected to the Rubin test for patency of the fallopian tubes by the intra uterine inject on of a r A few minutes after leaving the gynecolog st s office she experienced sharp pa n in the left upper abdomen with radiation to the neck At the same time there de eloped a paroxysm of tachycard a

She returned to the gynecologist s office where I sa the patient with him The present ing featu es were the complaint of pain and a heart rate of 15? per minute Reasoning that both pa n and tachycard a were caused by a bubble of air under the left half of the diaphragm we placed the patient flat on the table and elevated the foot end Not only d d the pain promptly stop but the h art rate fell in a few seconds to 76 Some minutes later when the patient assumed the e ect po ition both pain and tachycardia returned to be promptly rel eved when she as aga n inverted Some hours later when the air bubble had been absorbed she was free of symptoms

Eighteen months later she ent through her first pregnancy without trouble and was well when I st heard from n 1934

Obviously the paroxysm of tachycardia must have been mediated by a reflex mechanism

When an attack of paroxysmal tachycardia lasts long enough even though it was initiated by an allergic mechanism and in the absence of obvious cardiac disease then the exhaustion of even a normal heart might result from the prolonged excessive effort When a patient is seen for the first time in the latter stage the physician may well assume the presence of serious myocardial damage and may even overlook the paroxysmal and functional nature of the underlying tachycardia The next case illustrates this point

**Case 93** In December 19 6 I was called to see Mrs M V age 6 in consultation with her family phys cian For some years she had had h art trouble with increas ng breathlessness and palpitation on exert on so that her activ ties were more and more lim t d S x we ks before my vis t she had been forced to tak to her bed because of dyspn and ncreas ng per pheral ed ma Her doctor believed her to be in extremis from circulatory fail re probably based on an arteriosclerotic myocarditis The appearance of

the patient fully justified this view orthopnea cyanosis many rales at the lung bases a swollen liver considerable dependent edema The heart rate was about 200 The administration of digitalis over a period of the last two months had been of no avail

There was one item in the history however which in my opinion had not been given adequate consideration The earliest evidences of heart involvement were attacks of palpitation and tachycardia that had been occurring for at least 0 years, and long before breathlessness or any other sign of circulatory inefficiency appeared Moreover these attacks of tachycardia came on suddenly independent of exertion and they stopped as suddenly as they began A diagnosis of paroxysmal tachycardia was therefore ventured and quinidine treatment was begun at once

On the second day the heart rate fell suddenly to under 100 and the improvement in the clinical picture that followed was most striking For a time short paroxysms recurred several times a day but grew less frequent as a suitable daily ration of quinidine was reached On December 30 19 6 she was able to make a 1?5 mile motor trip to the home of friends in Philadelphia The next day an electrocardiographic study in the midst of an attack was made The cardiac rate in the upright position was 195 and in the recumbent position 185 The rhythm was regular On the next day January 1 19?7 the tracing showed a normal rate of 5 and a regular rhythm Dr Wolferth reported 'The tracings show beyond question that the tachycardia is of paroxysmal auricular form The negative T waves would point also to definite myocardial disease The patient soon learned to adjust her quinidine dosage to prevent more than an occasional short attack of tachycardia

Over four years later on April 17 1931 the patient having been fairly well in the interval another electrocardiogram was made It showed normal T waves At the time of writing (February 1940) the patient continues to be remarkably well in spite of her 80 years

**Discussion** This patient had had asthma in childhood ragweed hay fever every autumn for many years a rash each time she ate strawberries and digestive upsets whenever she ate certain foods Several members of her family had been asthmatic The avoidance of the foods causing indigestion also greatly reduced the frequency of the paroxysms of tachycardia so that she has gone as long as six months without an attack and without taking quinidine

## OTHER CONDITIONS

From here on it becomes difficult to enumerate additional cardiac conditions provoked by an allergic mechanism In most of those to be mentioned in this section the allergic mechanism has been suggested by some enthusiastic allergist but his view has not received confirmation The usual fallacy in the allergist s conclusion has been the assumption that because a cardiac symptom or disease became worse at the time of an allergic episode that allergy was directly responsible for such exacerbation The truth is that any sufficient overload allergic or otherwise can bring on symptoms in a diseased heart it is the overload that i responsible not the nature of the overload In a few instances an exceptional case of a condition may be on an allergic basis whereas the overwhelming majority have no such connection The double danger then is that the allergist assuming too much on the basis of his few cases makes unwarranted generalizations about the role of allergy in that condition while

the clinician who is not sufficiently allergy minded completely misses the occasional case caused by allergy occasional yet one in which treatment offers so much more than in most other kinds of heart disease

What follows is therefore an attempt both to curb the allergist's unwarranted enthusiasm, as well as to put the general practitioner on his guard for the occasionally allergic condition

### Paroxysmal Auricular Fibrillation

Not to be confused with ordinary auricular fibrillation this much rarer form of the arrhythmia has been attributed to various factors including toxic states (acute infections, anesthesia thyrotoxicosis poisoning by carbon monoxide, alcohol tobacco etc) gastro-intestinal disorders or even emotional upsets (page 394) A significant fact from the standpoint of a possible allergic mechanism is that the paroxysmal arrhythmia is at times the only abnormal cardiac finding

## ILLUSTRATIVE CASE

The following case is to my knowledge the first recorded instance of an allergic paroxysmal auricular fibrillation

**Case 94** Mrs A W white aged 61 has experienced characteristic attacks of palpitation for at least 5 years and probably longer The attacks used to come at intervals of weeks or months beginning suddenly lasting a matter of minutes and stopping rather more slowly than they began She first consulted Dr Leaman in 1930 but never presented herself during an attack An electrocardiogram in an interval showed a normal tracing In 1933 she survived an attack of Type 3 lobar pneumonia in the midst of which she had a paroxysm of fibrillation that was promptly controlled by quinidine Following the sudden death of her mother from coronary occlusion and the prolonged illness of her husband the patient experienced more frequent attacks that lasted up to several hours In one of these Dr Leaman secured an electrocardiogram that showed auricular fibrillation yet in the subsequent interval he found a normal tracing (Fig 144) During 1937 she had attacks at intervals of days or weeks but was able to control them promptly with quinidine Some of these paroxysms followed soon after a meal In September 1937 she was admitted to a hospital for study but all examinations including Roentgen investigation of gallbladder and gastro intestinal tract gave negative findings

In January 1938 Dr Leaman referred her to me for investigation of a possible sensitivity From the standpoint of allergy these points were highly significant as a child she had eczema and even recently she has had an itchy scaly rash back of an ear For many years she had typical and rather severe migraine A paternal uncle and her paternal grandmother had eczema and a paternal uncle had migraine A maternal uncle had hay fever Skin tests showed the patient to be sensitive to a number of food

In the two years since she was tested she has experienced far fewer and milder attacks and these have usually followed a break in her diet

### Extrasystoles

Rowe [32] Wirley [416] and others have reported the occurrence of extrasystoles in allergic patients after the eating of certain foods and their disappearance after avoidance of the specific excitant Here again although an allergic mechanism might be involved it seems more likely that the arrhythmia is provoked in other ways (vagus reflex toxic action etc)

cardiac condition are overlooked In others there is no mistaking the presence of both diseases Then there are those in whom the cardiac failure so dominates the picture that the co-existing asthma is not recognized

Asthma is a peculiarly serious complication for a patient with heart trouble for reasons too obvious to mention The recognition of the asthma in the cardiac patient is peculiarly important because the treatment of the asthma offers so much in the way of probable help not only for the asthma but indirectly for the heart by reducing its load Here then is a subject that should challenge the interest of every doctor who treats patients with heart disease

## Does Asthma Cause Heart Disease?

This question has given rise to more discussion among allergists than might be anticipated at first glance The answer to the question probably depends on a number of circumstances

Asthmatic symptoms occur in one of three ways (1) Short, clear-cut paroxysms lasting minutes or hours and with symptom free intervals whose length is determined by the time elapsing between intermittent contacts with an external cause (2) Continuous wheezing for weeks months or years often mild at times severe and usually with slight to severe paroxysmal exacerbations Continuous asthma may be caused by constant exposure to external causes or to a continuation of external causes and internal causes, notably infection with or without bacterial allergy (3) Status asthmaticus denotes an intensely severe and prolonged attack of dyspnea lasting days or even several weeks often with fever, usually with scanty viscid bronchial secretion that is hard to cough up and may produce bronchial obstruction atelectasis and even fatal asphyxia

**How Can Asthma Affect the Heart?** There is some experimental evidence that in the perfused heart of a sensitive animal there results a diminished coronary flow Criep[75] has shown that in man during an asthmatic paroxysm there are transient electrocardiographic changes that might be a result of such a reduction of blood supply This might lead to symptoms such as premature beats or possibly even angina Such paroxysmal asthma of itself, however, apparently does little or no permanent damage to the heart

Continuous asthma by reason both of its incessant annoyance and increased and continued exertion is undoubtedly an important factor in aiding and abetting other causes of heart disease of whatever nature they may be It probably is also responsible for some heart disease in that its most serious complication emphysema may lead to eventual myocardial trouble and failure (page 427) As yet there are not available a sufficient number of cases observed over many years to draw any statistical conclusions although Old[83] and Dublin and Marks[83] on the basis of life insurance data find a death rate among asthmatics that is 21 to 30 per cent higher than the normal This increased death rate is largely attributed by them to cardiac disease

Emphysema In regard to the development of serious emphysema in the course of asthma I would like to record two clinical impressions and one note of diagnostic caution One important factor in the etiology of emphysema in asthma appears to be an inherent quality of the pulmonary tissue the grade of the rubber" as Osler expressed it Of two asthmatic patients whose diseases appear to be identical the one may go for many years without developing emphysema whereas the other s lungs reach an advanced degree of emphysema in a year or so A second point that seems to be important in regard to the development of emphysema is that the shape of some chests is such that their owners have a shorter way to go than do others before their bony thorax has reached its limits of expansion It certainly seems to me that the asthmatic who begins with a thick stocky chest with ribs that approach nearer to the horizontal and with a deep antero-posterior diameter, makes poorer weather of his asthma than does the asthmatic whose chest at the start is rather longer with sloping ribs a cross section that is more oval than round and consequently showing wider variations in chest capacity at the extremes of the respiratory cycle

The diagnostic caution is this a diagnosis of emphysema should be made only with great hesitancy during the presence of frank asthmatic symptoms The roentgenologist is particularly prone to make this error when reading the films of an asthmatic patient yet films made a few minutes after the asthma has been relieved by epinephrine will be reported as normal A comparison of physical findings before and after epinephrine is always in order in such cases Vital capacity figures are then particularly helpful

### Diagnostic Problems

Many cases of co existence of asthma and heart disease in the same patient present no difficulties in this regard Pronounced asthma is easily recognized by the experienced clinician and the same may be said for cardiac disease There are however a number of situations in which diagnostic error may arise (page 115) Asthma especially when continuous may at times lead the clinician wrongly to diagnose a heart disease that does not exist Again the asthma may so dominate the picture that an associated cardiac lesion is overlooked In yet other patients the asthma is so overshadowed by the heart trouble as to be overlooked

## ILLUSTRATIVE CASE

Here is a case in which heart disease was wrongly suspected in a patient with severe asthma

**Case 96** Mrs C F a white woman 52 years old had had asthma since the age of 11 During the last two years the asthma had been practically continuous The least exertion caused distressing increase of dyspnea Because of nasal obstruction due to polyp she was brought to the office of a rhinologist The rhinologist was so impressed by her

breathlessness on exertion and her marked cyanosis that he promptly sent her to a cardiologist The cardiologist on the other hand finding no evidence of heart trouble either clinically or by electrocardiogram sent her to me for study of her asthma The asthma was greatly relieved by treatment according to allergic principles so that she proved a good subject for nasal surgery and eventually was restored to her usual health The cyanosis which had so strongly suggested heart disease to the rhinologist cleared up when she stopped taking a proprietary asthma medicine containing acetanilid

Severe asthma may for a time mask the development of cardiac disease This is more likely to occur in the elderly and in those with fairly continuous *rather than paroxysmal asthma In part, at least, this is a result of* the masking of physical signs in the heart by the overexpanded and noisy lungs The chance for diagnostic error will be lessened if the physician makes it customary to examine the heart at a time when epinephrine has minimized the asthma but when the pressor effects of the drug are wearing off

Asthma is not always a clearly defined condition with obvious symptoms and findings In its milder forms the patient experiences nothing beyond a tendency to bouts of coughing and a slight feeling of tightness in the chest Such symptoms may last only a few minutes after being provoked by exertion or hearty laughter Or they may be more prolonged and are then considered as colds, to which the patient casually refers as his 'cigarette cough or his 'bronchial trouble Physical examination would disclose a few wheezy rales at the height of symptoms but would be negative in the intervals Neither the patient nor his physician thinks of the condition as a true allergic asthma Yet such it is and at any time it may assume major proportions and serious significance especially when heart disease enters the picture Its recognition then becomes a matter of vital importance

## ILLUSTRATIVE CASES

Examples of this are furnished in the following abstracts of cases reported by me elsewhere [184]

**Case 97** C S a man aged 53 was admitted to the University Hospital because of dyspnea and edema of the feet and legs Dyspnea had begun two years before and gradually increased until he had an attack of bronchopneumonia 14 months before *admission Following this the breathlessness became much worse* at first on exertion then without effort especially toward nightfall Increasing palpitation and peripheral edema caused him to consult a physician who diagnosed heart trouble and gave him digitalis There was no personal or family history of asthma or any other allergic condition For 16 years he had worked in a very dusty occupation; during the last three *years he had been required to wear a mask*

PHYSICAL EXAMINATION showed obvious dsypnea cyanosis edema advanced emphysema a greatly enlarged heart with feeble sounds and a rapid rate a swollen liver and many musical wheezing rales in both lungs The roentgenogram showed enormous enlargement of the heart and probable pneumoconiosis The electrocardiogram gave evidence of severe myocardial trouble There was no eosinophilia

Discussion A diagnosis of advanced emphysema pneumoconiosis and severe myocardial disease with congestive heart failure seemed to offer an adequate explanation for this symptom picture But the wheezy character of the rales aroused the suspicion of allergy On being tested he was found highly sensitive to feathers Avoidance of feathers was followed by marked improvement for a month Then the signs of heart failure gradually returned and the patient finally died The wheezy rales however, did not recur Had the asthmatic component been recognized much sooner, it might have served to delay the eventual failure of the heart

Case 98 W P a man of 51 had complained for two years of cough and hoarseness later of increasing dyspnea worse on exertion and finally of substernal pain Stud es established a d agnosis of syphil tic aneurysm of the aorta and a paralysis of the left vocal cord There was no h story personal or familial of allergic disease and the blood showed no eosinophilia From time to time however there were wheezy rales in both lungs and it became evident that his aneurysmal symptoms were worse when the wheezy rales were present Skin tests d sclosed sensitivity to feathers wool and horse hair Avoidance of these substances resulted not only in disappearance of the rales but also in strik ng improvement of his aneursymal symptoms and undoubtedly prolonged his life He d ed nearly three years later when the aneurysm ruptured

Discussion The finding of musical rales in any patient suffering from heart disease should always raise the question of a co-existing asthma True as it is that not all that wheezes is asthma the fact remains that asthma is by far the most common cause of wheezy rales

At times the moist rales of heart failure may mask or completely replace the musical asthmatic rales The failure to obtain positive skin tests although in itself not an uncommon finding in asthmatics of advanced years still further lulls to rest the clinician s suspicion of an asthmatic condition Under these circumstances the therapeutic effect of epinephrine on the patient s breathlessness may be of diagnostic help The following is an example

Case 99 W S a man aged 65 was admitted to the hospital with chief complaints of dyspnea cough and palpitation For 40 years he had had a chronic cough worse in winter At 48 he had h s first attacks of nocturnal dyspnea, wh ch then recurred several times a year usually after catching cold More recently the dyspnea had become less paroxysmal but more or less constant and usually related to exertion or to certain cardiac phenomena For 20 years he had had attacks of fluttering of the heart with tachycard a palpitation precordial distress and in the last few months typical ang na For several years he had known that he had a high blood pressure There was no fam ly hi tory of allergy

Physical examination showed a drowsy orthopneic patient cyanotic and with Cheyne Stokes b eathing The chest was very emphysematous and there were numerous mo st rales at both lung bases The he rt was definitely enlarged (transverse diameter by orthodiagram was 16 8 cm) the sounds were feeble the rate rapid and there were many extrasystoles The blood pressure was elevated The live was felt 8 cm below the ribs in the midclavicular l ne a d was d stinctly tender There was no edema Th blood urea nitrogen was 4 mg per 100 cc The roentgenogram of the chest showed the enlarged heart and marked passive congestion of the lungs The electrocard og am gave evidence of severe myocardial d sease In view of the old history of asthma skin tests were made but with negative results The whole p cture then seemed to be one of

hypertensive cardiovascular disease with rather acute myocardial failure Venesection and rapid digitalization were discussed by the house officers

However in view of the old asthmatic history the attending physician suggested the repeated use of small doses of epinephrine (3 minims) as the first therapeutic measure There resulted marked and rapid improvement in all symptoms In five days the signs of cardiac decompensation had nearly subsided the pulse became normal as did the blood urea nitrogen After four weeks he walked out of the hospital

**Cardiac Asthma** At this point I wish to refer briefly to the syndrome cardiac asthma discussed more fully elsewhere (page 301) It is applied to dyspnea that is purely of cardiac origin is attended by rather sudden pulmonary congestion and edema and so has a distinctly paroxysmal manner of occurrence This condition when produced in the manner described is in no way related to allergic asthma In fact the use of the word asthma in this instance is unfortunate because it leads to unnecessary confusion in the minds of students It must be remembered however that the word asthma in Greek has simply the significance of breathlessness and is therefore practically synonymous with dyspnea Today *asthma is restricted to a type of dyspnea due to the* bronchospasm and mucosal edema of an allergic reaction

**Paroxysmal pulmonary edema** (moist not wheezing rales) may result in occasional instances when an asthmatic with severe cardiac disease is exposed to the things to which he is sensitive Vaughn[379] has directly observed two such cases the excitants being inhaled orris root in one and ingested egg in the other Swineford[371] even suggests that a majority of cardiacs who develop cardiac asthma are allergic In my patient W S cited in case 99, the findings were consistent with a diagnosis of cardiac asthma yet the relief obtained through epinephrine strongly suggests the allergic nature of the attack

It would therefore be wise in every case of cardiac asthma to think routinely of the possibility of an allergic factor in its production

## Principles of Diagnosis

The diagnosis of asthma in cardiac patients differs in no wise from that in other individuals The prime requisite however is an alertness on the part of the physician attending cardiac patients as to the possibility of asthma Especially is such alertness called for when the cardiac phenomena completely dominate the clinical picture as they so frequently do It must always be remembered that probably one in every 75 individuals has or has had asthma and that one in every seven is allergic

A detailed discussion of the diagnosis of asthma is out of place in this text It will be well however to outline the principles involved

**Evidence of Allergy** The possibility that a patient is allergic may promptly be suggested by the fact that he has presented at some time in his life a number of symptoms that may easily be recognized as allergic. He may have had frankly paroxysmal dyspnea in attacks that occurred whenever he was exposed to certain things and that were relieved by

epinephrine or ephedrine Other such obviously allergic conditions include hay fever perennial allergic rhinitis (paroxysmal excessive sneezing is as a rule allergic) urticaria eczema food or drug idiosyncrasy commonly also migraine A family history of these conditions further suggests the possibility that the patient himself may have inherited the allergic characteristics

The important thing from the standpoint of the history is that questioning along allergic lines must invariably be a part of the routine of all history taking

Whenever the dyspnea tends to come in paroxysms in a cardiac patient there is an increased possibility that an allergic factor may be involved This is even more true when the paroxysms occur independently of exertion or when they tend to recur at certain times and places

The finding of wheezing rales in the lungs should always raise the possibility of an allergic cause in their production This possibility becomes a decided probability if epinephrine promptly clears up such rales

Having decided on the basis of the foregoing criteria that the patient probably has asthma as well as cardiac disease one has made only the first step in arriving at a complete allergic diagnosis To arrive at such a complete diagnosis requires

(1) **A searching history** that includes all details of his contacts with substances to which he might be sensitive

(2) **A physical examination** which must always include an examination of the upper respiratory tract by a rhinologist using the nasopharyngoscope and transillumination of the sinuses In this way anatomic defects such as a marked deflection of the nasal septum with firm septoturbinal contacts or secondary pathologic changes such as sinus infection and mucous polyps will be found In selected cases subject of course to the limitations imposed by the cardiac disease bronchoscopy is indicated to discover bronchiectasis or bronchostenosis

(3) **Laboratory studies** including roentgenograms usually of the sinuses often also of the chest and in some instances of the bronchial tree after lipiodol instillation

(4) **Complete skin tests** by some one who knows what to test for and how to interpret the results

(5) **Treatment itself is diagnostically important** for it determines not only the significance of the results of the skin tests but may serve to discover causes which the tests failed to find

(6) **Subsequent Studies** Such a complete diagnostic study is not necessarily the last diagnostic survey The fundamental trouble with the allergic patient is not that he has asthma or that he is sensitive to horse hair but that he has inherited the ability to become sensitized to things in his environment more easily than do normal persons Therefore his sensitization pattern is not a fixed and static thing but can and does change in the course of the years old sensitivities disappearing and new ones developing This possibility must therefore be considered when the patient suffers a relapse

### Principles of Treatment

Here again there is no fundamental difference in the management of cardiac patients with asthma as compared with asthmatics who have no cardiac disease There are, to be sure occasional instances in which drug therapy of one condition needs to be modified in consideration of the other The chief of these will be mentioned later

I shall confine myself to a brief outline of therapeutic principles For details as to my views on the subject the reader is referred elsewhere [187]

**Allergic Factors** The first axiom in allergy is that avoidance of that to which the patient is sensitive gives by far the best therapeutic result But to be wholly effective such avoidance must be complete When complete avoidance is impossible or highly impractical (e g house dust or pollen) then one should strive to attain a partial avoidance as great as may be The production of symptoms by an allergen in a sensitive patient is a quantitative matter A very little of the allergen may cause no symptoms at all More allergen will provoke mild symptoms and much allergen will cause serious symptoms The more of the allergen which the patient avoids the more likely is he to be helped by the next therapeutic measure to be applied desensitization by injection of extracts of the things that cause his asthma Sensitivity to pollens and to house dust most commonly calls for such injection treatment In addition to these orris root feathers hair and an occasional occupational dust may call for desensitization when unusual circumstances make their complete avoidance by the patient impossible Food sensitivity calls for desensitization as a rule only in case of the foods hardest to avoid egg milk and wheat and then this is attempted by feeding (rather than by injection) of increasing amounts beginning with quantities too small to produce symptoms

**The Treatment of Complications** This calls for some consideration of the treatment of the cardiac disease From the standpoint of drug therapy attention is called at this time to two things that should be remembered in the case of allergic cardiac patients Allergic patients furnish the bulk of those who have drug idiosyncrasies These include such drugs as quinine, quinidine salicylates iodides barbiturates and opiates When a patient gives a clear-cut history that he is sensitive to a drug it should never be given except under the greatest need and with the greatest precautions lest the result prove dangerous The second point of warning from the angle of cardiac therapy is that opiates and especially opiates together with atropine drugs so useful in cardiac disease must be used with the greatest caution in patients with intense asthma and greatly reduced and very viscid sputum that is in status asthmaticus Their use in status asthmaticus has been the most common cause for death in asthma

Nasal Complications often call for surgery provided the cardiac condition permits There may be need for a submucous resection the drainage of infected sinuses or the removal of nasal polyps Such operations should

be performed under local anesthesia They should not be done during a pollen season lest the patient develop a new pollen sensitivity

THE TREATMENT OF INFECTION in sinuses or bronchial tree requires careful and sustained treatment In addition to surgical measures to insure drainage of sinuses this includes the use of vaccines autogenous or stock Such vaccine therapy must be very cautiously used especially from the standpoint of dosage lest injections be followed by serious reactions in those sensitive to bacteria Initial doses should never exceed two million organisms and may have to be much smaller Injections are best given at intervals of not less than one week Change of climate especially during the winter months is a valuable measure when feasible

**The Treatment of the Asthmatic Dyspnea** The most effective drug in asthma is of course epinephrine It may be given by inhalation of a vaporized 1 100 solution or the subcutaneous injection of 1 1000 solution Here the doses should be small 2 or 3 minims repeated as often as needed rather than larger doses Epinephrine may be suspended in oil (1 500) or dissolved in gelatin that is solid at body temperature but liquid at a slightly higher temperature The absorption of the drug is thereby delayed and its effect greatly prolonged In the presence of cardiac disease however the use of epinephrine has certain obvious limitations even contraindications These include coronary disease and hypertension in which such a violently acting pressor substance should be used with extreme caution or not at all Less effective and also less dangerous are ephedrine neosynephrine and related drugs These drugs while less useful than epinephrine in severe asthma, are often very helpful in preventing attacks when they are routinely administered several times a day Here again smaller doses e g 1/8 grain to 3/8 grain of ephedrine sulphate should be used to avoid unfavorable effects on heart and blood pressure

IODIDES are of great value in cases of chronic and more or less continuous asthma Small doses 2 or 3 grains t i d should be given by mouth and over long periods

ANTIPYRIN AND ACETANILIDE Some asthmatics have found that they get considerable relief through certain proprietary mixtures containing these two drugs Their use in cardiac patients is to be discouraged because of the danger of methemoglobin formation and consequent impairment of respiratory function

NONSPECIFIC PROTEIN SHOCK THERAPY so useful at times in chronic asthma is contraindicated in the presence of cardiovascular disease notably myocardial weakness hypertension and arteriosclerosis

In more severe attacks *aminophyllin* 2 grains by mouth or far better 7½ grains in 2 cc of solution injected intravenously may relieve when epinephrine fails

IN THE MOST SEVERE ATTACKS *status asthmaticus* one is dealing with a major emergency which calls for prompt and decisive action The first indication is immediate hospitalization if at all possible The patient should be in a room alone not in a ward Bedding and all contents of the room

must be in keeping with his known allergic pattern This must be arranged in advance On several occasions I have saved a patient's life by taking out of his oxygen tent the feather pillow that was choking him to death

OXYGEN OR OXYGEN WITH HELIUM, is the most effective means of combating the cyanosis

ANESTHETICS At times surgical anesthesia has proved life saving I prefer to use avertin, 60 to 80 mg per kilo of body weight given in solution by bowel

VENESECTION in the presence of pulmonary edema is of recognized value One should remember the principle set forth by the old phlebotomists that 10 ounces rapidly withdrawn was more helpful than 20 ounces removed slowly

**Climatic Treatment** There is no climatic treatment of asthma There is only climatic treatment for the individual asthmatic What may be a favorable climate for one may not prove so for another A few guides in selecting a climate may be ventured but without any advance guarantee of efficacy An equable climate without severe storms is preferable for most asthmatics The asthmatic with cardiac disease should never go to an altitude higher than 2000 feet Those with much bronchitis and considerable sputum will be better off in the dry climate of southern Arizona or southern California On the other hand if the patient's sputum is scant and viscid a moist climate and near sea level is better (Georgia Florida Gulf Coast) High humidity and especially fog are badly tolerated by most asthmatics Whenever a climate change is contemplated care must be taken that the patient does not encounter in his new environment the things to which he is sensitive

From what has been said it is clear that the management of a patient with asthma and cardiac disease requires of the physician a degree of skill and judgment not exceeded in the realm of clinical medicine

# 17

# CARDIAC PROBLEMS IN SURGICAL PRACTICE

## THE SURGICAL RISK

We have advanced considerably since the days when auscultation at the last moment in the anesthetizing room was considered sufficient to determine the risk from the cardiac standpoint Today the functional capacity of the circulation is stressed while the murmurs that attend valvular lesions carry far less weight in the final estimate of the patient s ability to stand the added strain of anesthetic and operation There is also a greater realization today that the circulation is not the simple problem in hydraulics it was once believed to be and the heart is not the only motive organ

**The role of the peripheral circulation** in the production of many symptoms in surgical patients that were formerly regarded as cardiac manifestations is now appreciated A better understanding of the signs and symptoms that accompany surgical shock aids in the recognition of this condition and still further reduces the number of heart deaths reported by surgical services

**A detailed study** of every aspect of the cardiac patient is an essential prelude in the assessment of the surgical risk Here again the classification of the American Heart Association (page 59) proves most useful in assembling the essential details for only when the cardiac diagnosis is summarized under etiology anatomy physiology and functional capacity is the way paved for consideration of the problem of the surgical risk I have found the small form shown in Table XI a handy guide in these cases If filled out by the surgeon and internist before the consultation it will sharply outline the situation and save considerable time

Response to Exercise A knowledge of the heart s response to the exercise imposed by daily activities will aid in giving an opinion as to its probable conduct during anesthesia and operation Many times the burden imposed by operation is far less than this accustomed load Consequently if the patient tells us that he has no dyspnea or chest pain on ordinary exertion we can be reasonably sure that the functional capacity of the heart will not be dangerously taxed by the operation The few exceptions to this rule are the usual unpredictable types coronary disease with angina and syphilitic aortitis Sudden death of patients in these groups may occur during surgical procedures just the same as it may occur during the ordinary activities of every day life but fortunately this is a rare event

TABLE XI

PRE-OPERATIVE CARDIOVASCULAR CONSULTATION SHEET

*Name* | *Age* | *Service of*

Occupation
Surgical diagnosis
Operation contemplated
Past medical history (resume)
Family history of cardiac disease

THE CARDIOVASCULAR STATUS

Symptoms

Dyspnea or chest pain (check)— { On level / On grade / On stairs / On hurrying / Against the wind }

Cardiac pain— { Location / Duration / Radiation }

Duration of above symptoms?
Are they stationary or progressive?
Is cardiac failure present? | Edema? | Its extent

EXAMINATION OF THE CARDIOVASCULAR SYSTEM

General appearance
Blood pressure Systolic | Diastolic
Venous pressure
Peripheral vessels (check) Barely palpable | Thickened
Tortuous | Calcified
Retinal vessels (if examined)
Heart
Size (record position of apex beat if palpable)
Thrill (location and time)
Heart sounds (intensity splitting)
The rhythm
Murmurs (location transmission in erect position and in recumbency)
Response to previous operations
Response to exercise test
Result of electrocardiographic study
Result of chest roentgenogram or fluoroscopy
Orthodiagram

Cardiovascular diagnosis (A) Etiologic
(B) Anatomic
(C) Physiologic
(D) Functional capacity
Therapeutic Class

The grade of risk
Result of operation
Cardiovascular condition on discharge
Cardiovascular prognosis

Patients who have complete heart block should be judged on the basis of the myocardial reserve Usually they cannot carry on ordinary activitie of life without discomfort and are poor risks Well compensated rheumatic and hypertensive hearts usually stand anesthesia and operation very well When rales are present in the lung bases or other signs of congestive failure can be demonstrated the patient is a poor risk and should not be operated on until the congestion clears, and then only if the situation

without operation is an immediate threat to life itself An estimation of the grade of cardiac risk based on a study of the patient s functional capacity may be obtained by the application of the data summarized in Table XII

TABLE XII

THE GRADE OF RISK BASED ON A STUDY OF THE FUNCTIONAL CAPACITY

| | | |
|---|---|---|
| Grade I | Class 1 of American Heart Association (New classification) | Good risk Here are included patients in whom ordinary physical activity does not cause undue fatigue palpitation dyspnea or chest pain all cases of well compensated valvular heart disease except syphilitic hypertensive heart disease with no renal involvement |
| Grade II | Classes 2 and 3 of American Heart Ass ciation | Risk fair with careful medical supervision and pre operative treatment Emergency surgery only until risk improved Here are grouped cases of beginning congestive failure and angina The e cases all show undue fatigue palpitation dysp nea or chest pain on *ordinary* physical activity |
| Grade III | Cl ss 4 of American Heart Association | Surgery contraindicated These patients show signs of cardiac insufficiency at rest or signs of heart infection They cannot carry on *any* physi cal activity without discomfort Here are in cluded severe angina patients cases of cardiac decompensation with edema and severe dyspnea and patients with marked reduction of the myo cardial reserve |

**Type of Surgical Procedure** Finally, an estimation of the surgeon s ability in performing the contemplated operation and the probable duration of the procedure must be considered Certainly the patient who is on the operating table for three hours is not as good a candidate for recovery as the patient who is back in his room in half this time following the same operation provided an equal delicacy of tissue manipulation has been shown

## THE VALUE OF THE ELECTROCARDIOGRAM

The electrocardiogram is rarely the sole basis for estimating the surgical risk except in the presence of unusual arrhythmias or when coronary occlu sion is suspected However the electrocardiogram may be of indirect as sistance in evaluating the cardiac prognosis which will aid in making the decision regarding operation Many operations of convenience like hernior rhaphy and hemorrhoidectomy are not carried out in cases where the electrocardiographic findings point to a very serious lesion that is usually not compatible with more than a year or two of life The electrocardiogram may also ease the surgeon s worries by identifying a number of cardiac arrnythmias as harmless types THE ELECTROCARDIOGRAM IN ITSELF OFFERS NO SHORT CUT TO AN ESTIMATION OF THE SURGICAL RISK It is only a part of the cardiac examination and should be considered with the rest of the clinical and laboratory data in making the evaluation (Table XI)

Occasionally the electrocardiogram may be a great assistance to the sur geon in differentiating coronary occlusion from acute abdominal emergen cies when the pain is referred to the abdomen (page 479)

### Choice of Anesthetic

If care is used in the choice of the anesthetic and the anesthetist no untoward effects on the heart need be feared Although disturbances of cardiac rhythm during induction as well as fall in the blood pressure and cyanosis are often noted we cannot blame the heart for what is often the result of a poorly administered anesthetic If a sudden increase in the pulse rate that attends the onset of a paroxysm of tachycardia occurs during anesthesia, this should not be a cause for alarm if dyspnea and venous stasis are absent Vagus pressure often stops the attack, and it is rarely necessary to resort to emergency measures (page 383)

Electrocardiographic studies made during operation on 109 patients by Kurtz and his coworkers[171] showed marked changes in the rhythm Sinus arrhythmia extrasystoles, downward displacement of the pacemaker were the most common alterations although paroxysmal auricular fibrillation and heart block were recorded in rare instances Chloroform was found to produce the greatest number of irregularities and procaine the least The association of chloroform with instances of ventricular fibrillation has likewise seemed to decrease the frequency of its use in modern surgical practice

Care should be exercised in the use of epinephrine combined with the various preparations used in local anesthesia in patients with coronary disease and angina since instances of coronary occlusion following operation have been traced to this source Epinephrine may also provoke ventricular fibrillation in susceptible cases if not used cautiously

Nitrous oxide oxygen if skillfully given is satisfactory for cardiac patients However if the situation is not in the hands of a well trained anesthetist ether, using the open drop method, is safer

### CARDIAC ARREST DURING ANESTHESIA

The sudden stoppage of a normal heart during the administration of an anesthetic is an emergency that has a happy ending in few instances Considering the advances made in the experimental laboratory and the recent successful defibrillation of the human ventricles by Beck prompt action by previously trained resuscitation squads should bring success in a much higher percentage of cases

**Ventricular fibrillation and cardiac standstill** are the terminal mechanisms that must be combated In 1850 Hoffa and Ludwig first showed that electrical stimulation of the mammalian heart produced ventricular fibrillation and death Provost and Battelli, in 1899 succeeded in applying a countershock directly to the fibrillating heart by placing electrodes on its surface and bringing about complete standstill Hosler Kouwenhoven and Langworthy in 1933 demonstrated that adrenalin and calcium chloride after the countershock and standstill restored the normal beat Wiggers used massage before applying the countershock

and has been successful in re establishing normal rhythm in dogs with fibrillating ventricles

Mautz emphasizes the importance of adequate pulmonary ventilation while attempts are being made to revive the fibrillating ventricles A supply of oxygen can be delivered to the lungs by the use of a face mask or an intratracheal tube Direct massage of the heart moves the oxygen from the lungs to the brain tissues where the lack of oxygen produces irreparable damage in three to five minutes A chest incision with removal of one or two cartilages should be quickly done and the heart grasped in the hand In this manner the heart may be emptied and circulation continued

**The process of defibrillation** should next be attempted Mautz working in Beck s laboratory has demonstrated that procaine hydrochloride is useful in the defibrillation process by reducing the irritability of the heart Two cc of a 5 per cent solution are injected into the right ventricular cavity Massage of the heart forces the drug into the coronary circulation and the cardiac tone is quickly reduced When an electric current of 1 0 to 1 5 amperes is then sent through the heart by placing two electrodes directly on its surface the fibrillation is usually replaced by standstill At this stage 1 cc of a 1 to 1000 solution of epinephrine hydrochloride well diluted and added to a 5 per cent solution of calcium chloride is injected into the right ventricle and massage continued Co-ordinated contractions of the heart are now initiated in many cases although small subsequent doses of epinephrine may be necessary To guard against dilatation the heart should be carefully watched for 20 minutes before the chest is closed A team possessing the knowledge of the essential apparatus and the technic to employ in using the drugs that have been recommended by Beck and his associates in the defibrillation of the human ventricles should be available to every surgical service

**Other Procedures** In the absence of the team and equipment necessary to defibrillate the ventricles either of two procedures may be employed The heart may be massaged through the diaphragm and in this *manner a flow of blood to vital cerebral centers* maintained or intracardiac injections may be used The combination of these two methods is more apt to be successful but the results will not equal application of the more accurate measures outlined above

Drugs Any of the numerous drugs that have been recommended for cardiac injection may suffice in cases of standstill if they are capable of producing an irritable ectopic focus at the site of injection [168] In the first stage of anoxemia there is a failure of impulse formation at the S-A node The piercing of the heart muscle itself is a stimulation and is followed by a cardiac contraction and the re establishment of normal rhythm In a later stage of anoxemia when the heart muscle is pierced by the needle it sends out a series of stimuli cau ing a paroxysm of tachycardia which is of the ventricular type if the mu cle of the ventricle is pierced or a paroxysm of auricular flutter if an auricular area is injected Hyman[169 170] points out that the important difference between these mechanisms in the second

stage of anoxemia is that paroxysms of ventricular tachycardia or flutter nearly always end in fibrillation and death, while paroxysms of auricular flutter lend themselves to treatment CONSEQUENTLY, IT IS FAR BETTER TO INJECT THE AURICLES IN AN EMERGENCY The right auricle is preferable and is just as accessible as the ventricular site The right auricle is entered by inserting the needle in either the third or fourth intercostal space just to the right of the sternum

## CAN THE SURGICAL RISK BE IMPROVED?

Co-operation of the surgeon and internist makes possible the improvement in the risk by proper pre-operative treatment in many cases Early congestive failure calls for delay in operation until it is controlled by the usual measures of rest and digitalis Recent studies also show the practical importance of biochemical considerations in improving the surgical risk Hypochloremia hypoproteinemia, as well as vitamin B deficiency (Chapter 21) influence edema in these patients and may increase the hazard of operation regardless of the technical skill of the operator The same rule applies to the delivery of patients in obstetric practice

Pre-operative digitalization is indicated only in the presence of congestive failure or auricular fibrillation Some surgeons still prescribe digitalis routinely before operation in all cardiac cases even though the patient shows no circulatory symptoms in carrying out the ordinary activities of life If this evidence of good circulation is present, how can it be further improved by the use of digitalis? The belief still exists that digitalis is useful in sustaining blood pressure and in increasing myocardial tone ' This it does not do, neither does it prevent hypostatic pneumonia in old people following operation On the other hand there is evidence that digitalis administered routinely before operation in the absence of cardiac failure may contribute to a fatal cardiac accident (page 86)

## POSTOPERATIVE CARDIAC ACCIDENTS

In patients who have cardiac disease of the coronary type with angina the possibility of an accident under the surgeon s care is the same as it is in the medical ward However, if convalescence from operation is complicated by sepsis or pneumonia these factors may precipitate an occlusion When surgery is imperative in the midst of an attack of coronary thrombosis, which is fortunately rare, the mortality rate will be high Sudden death following major surgery in patients who have syphilitic aortitis cannot be prevented

Another cause of death during the postoperative period is congestive failure Embolic and cerebral accidents occur more frequently in these patients with impaired circulation As a general rule all cardiac cases react badly in the presence of postoperative infection or pneumonia Congestive failure can be made an insignificant cause of postoperative deaths only when recognized and promptly treated before operation

## CIRCULATORY PROBLEMS OF SURGICAL IMPORTANCE IN THE DIAGNOSIS OF ABDOMINAL LESIONS

Abdominal symptoms may be produced by almost any of the etiologic types of heart disease previously discussed (Table XIII) The mechanism of the production of the symptoms however is variable For example the failure of the heart as a pump causing congestion of the abdominal organs is usually first reflected in enlargement of the liver Rheumatic heart disease with mitral stenosis is present in over 50 per cent of these cases although hypertensive or arteriosclerotic heart disease may be the cause Cardiac failure may produce the symptoms usually encountered in gastrointestinal-tract disease—gas anorexia nausea vomiting diarrhea fulness

TABLE XIII

CLASSIFICATION OF CIRCULATORY DISTURBANCES OCCASIONALLY PRODUCING ACUTE ABDOMINAL SYMPTOMS

(1) Cardiac Failure
- (A) Congestive Type
  - Liver Enlargement (right upper quadrant pain)
  - G I Tract Congestion (nausea vomiting gas hemorrhage etc)
- (B) Coronary Type (reflex)
  - Angina (pain referred to upper abdomen)
  - Coronary Occlusion (similar mechanism)

(2) Pericarditis
- (A) Acute Pericarditis (pain at times referred to abdomen)
- (B) Calcific Pericarditis (cardiac compress on) Ascites may be an early symptom

(3) Embolism and Thrombosis
- (A) Mitral Stenosis ( uricular fibrillation) Emboli from large left auricle to splenic renal superior and inferior mesenteric arteries and to bifurcation of aorta and iliacs
- (B) Subacute Bacterial Endocard tis Infected emboli from left s de of the heart Same locations as above

(4) Organic Vascular Changes
- (A) Aort c Aneurysm
  - (a) Symptoms produced by tumor growth
    - Displ cement of o g ns
    - Vertebral erosion
  - (b) Symptoms produced by rupture or d ssection
- (B) Arteriosclerosis Spasm (abdom nal angina)
  - Hemorrhage (in hypertension)
  - Thrombosis
- (C) Periarteritis nod sa.

and pain in the abdomen and loss of weight Mild yet clinically detectable degrees of jaundice may be added to the symptom of right upper quadrant pain caused by sudden distention of the liver capsule completing the masquerade[314] Ascitic fluid in small amounts may arise further to complicate the picture In some cases if constipation is present with the congestive failure this distention of the colon may reflexly inhibit the flow of bile from the liver as well as cause hypertonicity of the sphincter of Oddi Biliary dyskinesia follows[134]

Compression of the heart in calcific pericarditis by the decrease in dias

tolic filling may produce signs of venous engorgement, and the large liver and ascites may first attract attention to the condition (page 181)

**Embolism** In certain types of heart disease, embolism is a frequent mode of production of confusing abdominal symptoms Again patients with mitral stenosis and enlargement of the left auricle constitute the majority of this group Subacute bacterial endocarditis, with its tendency to involvement of the left side of the heart, is likewise a dangerous threat to the integrity of the arterial circulation below the diaphragm Emboli in vessels supplying the abdominal organs may be responsible for the sudden onset of symptoms simulating a variety of surgical lesions Occlusion of the mesenteric vessels presents a picture often diagnosed intestinal obstruction while a diagnosis of renal calculus may be made when an embolus lodges in the kidney

**Referred pain** from coronary-artery disease with angina or more frequently occlusion may simulate any type of gastro intestinal tract disturbance The abdominal reference of pain in cases of angina and occlusion, and infrequently in cases of acute pericarditis may simulate gall bladder disease ulcer or acute pancreatitis Similarly, reflexes from these organs may at times produce changes in the cardiac rhythm and even in the form of the electrocardiogram Recent experiments upon dogs by Owen[79f] and Crittenden and Ivy[79] have served to prove that this mechanism is more than a mere possibility Stimulation of the vagus nerve by disturbances in the gallbladder is no doubt the fundamental cause

**Palpable abdominal masses** caused by aneurysmal dilatations of the abdominal aorta particularly when they occur in women, are puzzling to the surgeon Rupture or dissection of the aneurysm produces an acute picture rarely correctly diagnosed and often leading to an unnecessary celiotomy Smaller hemorrhages although productive of the same degree of confusion may occur in abdominal organs in hypertension Arteriosclerosis of the abdominal vessels may lead to thrombosis and the clinical picture will depend upon the size and location of the vessels involved Reduction of the blood supply in the absence of thrombotic occlusion may give rise to abdominal symptoms that have been grouped under the clinical heading of abdominal angina Lack of proper blood supply (ischemia) when the demand on the gastro intestinal tract is greatest (after meals) produces pain

## ILLUSTRATIVE CASES

### Coronary Heart Disease Complicated by Acute Coronary Occlusion with Abdominal Reference of Pain

**Case 100** Mr J M., a mechanic age 50 was admitted to the College Hospital July 6 1938 with chief complaint of sudden severe epigastric pain coming on while at work He gave a history of two years treatment for gastric ulcer No roentgen ray study however had been mad

Physical Examination The patient showed marked pallor and was sweating p o

fusely Pulse 100 temperature 97 F and respiratory rate 30 The heart was not enlarged B P 110/70 There was upper abdominal tenderness but no rigidity

LABORATORY DATA W B C 12 000 The first electrocardiogram was negative A second electrocardiogram taken a few hours later showed characteristic T wave changes of coronary occlusion

**Discussion** The previous digestive tract disturbances complained of by this patient were no doubt secondary to the coronary lesion He suffered from a vague type of indigestion for some years and although he was never fully studied to prove the diagnosis of peptic ulcer it was assumed that this lesion was present because of the favorable effect of alkali therapy The history of this form of treatment centered the attention of the receiving ward physician on the gastro-intestinal tract the diagnosis of ruptured peptic ulcer was made and the patient was placed on a surgical service It is a matter of additional interest in this case that the usual leads of the first electrocardiogram were entirely negative and the typical electrocardiographic pattern did not appear until 18 hours after the onset of the attack At the same time a friction rub was heard over the precordium clinching the diagnosis It appears that the dangerous period for these cases in which there is abdominal reference of pain is the silent interval between the onset of the attack and the development of the typical electrocardiographic signs

### Coronary Sclerosis Followed by Acute Coronary Occlusion Diagnosed Gallbladder Disease

**Case 101** N M a male clerk of 51 when first examined complained of indigestion gas and occasional pain in the upper abdomen Dyspnea and palpitation had been present for years No edema or chest pain The significant findings were obesity slight increase in blood pressure (164/94) occasional premature beats but not much increase in heart size The electrocardiogram (Fig 154 A) showed only a left axis deviation The patient was placed on a dietary regime and told to make arrangements for a gallbladder roentgenologic study This he failed to do and when next seen 15 months later he showed no improvement The indigestion was worse the dyspnea was increasing and he stated that a week before the second examination he had experienced a very severe attack of indigestion This attack came on at night awakened him from his sleep and required a hypodermic injection of morphine for relief Another electrocardiogram (Fig 154 B) showed the presence of a recent coronary occlusion of the posterior type

**Discussion** Many times in patients of this age and build gallbladder disease and coronary disease coexist This association has led to a great deal of discussion and much speculation in the literature The exact relationship between a diseased gallbladder and the heart remains a complex subject although electrocardiographic evidence of improvement has been observed to follow cholecystectomy as will be shown presently Many investigators have also demonstrated the existence of important reflex pathways between the gallbladder and the heart Even effects upon the cardiac rate and rhythm have been shown to occur at the time of operation It has been my impression in a number of cases where striking postoperative improvement in the pain has been observed that the initial opinion as to the degree

of coronary involvement was incorrect In other words, most of the symptoms were produced by disease in a high lying gallbladder

If this is true our chief concern therefore lies in the correct identification of the major lesion The pain of acute coronary occlusion is often confused with the pain produced by gallstones and a celiotomy performed [10, 11] Pain, vomiting, fever leukocytosis are commonly met with in both conditions However, if the past history is carefully reviewed the patient with gallbladder disease will usually show a history of indigestion, while the

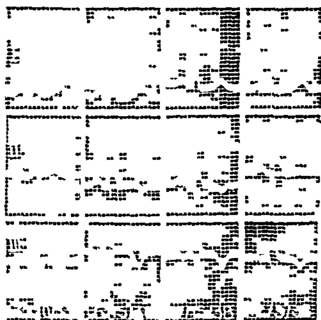


FIG 154 The electrocardiogram 3/20/37 shows left axis deviation 6/15/38 shows deep Q 3 and domed RS-T Intervals in leads and 3 (posterior coronary occlusion) The subsequent studies show a return of tracing toward normal Q 2 and Q 3 persist

patient with coronary occlusion may give a history of mild attacks of angina on effort A complete cardiac study, using the form suggested (see Table XI) will seldom fail to throw light on the correct diagnosis The degree of shock usually observed in coronary occlusion is not present in gallbladder disease nor is the sharp drop in the blood pressure so constant in gall bladder colic as it is in coronary occlusion The appearance of a friction rub over the precordium clinches the diagnosis in favor of anterior occlusion while the appearance of jaundice swings the balance toward gall bladder disease

Cholelithiasis may cause certain changes in the rhythm of the heart reflexly through the autonomic nervous system Inhibition of the heart can also be caused by stimulation of the filaments of the vagus arising from the wall of the gallbladder Heart block relieved by atropine has been reported

to result from gallbladder stimulation The jaundice produced in some patients by gallbladder disease may affect the cardiac mechanism in some instances

BILIARY TRACT DISEASE AND CORONARY DISEASE—MARKED IMPROVEMENT IN CARDIAC STATUS FOLLOWING CHOLECYSTECTOMY (DR I S RAVDIN)

Case 102* H B male age 45 when first examined on 4/1/35 gave a history of several attacks of typical biliary colic over a period of 18 months For approximately three years before admission there was in addition a history of dyspnea and a sense of substernal oppression following moderate exertion A roentgen study revealed the presence of gall stones and the electrocardiogram showed a flat T wave in lead 1 (Fig 155 A) A cholecystectomy was performed on 6/15/35 Convalescence was smooth and uneventful Eight months later the patient reported that he was in an excellent state of health Both cardiac and biliary tract symptoms had entirely disappeared This improvement was reflected in the electrocardiogram taken at this time which showed an upright T wave in lead 1 (Fig 155 B)

Case 103 Mrs B S an American housewife of 56 was well until nine months before admission At this time she had her first attack of epigastric and substernal pain A diagnosis of calculous cholecystitis was made and during the next six months she had a good deal of distention flatulence and heartburn At the end of this period there occurred another severe attack of epigastric pain again followed by several months of marked indigestion The patient lost 20 pounds in weight and could only be kept comfortable on a liquid diet Cholecystogram revealed abnormal function with multiple stones in the gallbladder An electrocardiogram (Fig 156 A) made on 3/1/36 showed slurring of the QRS complex and inversion of the T waves in leads 2 and 3 which was interpreted as evidence of myocardial damage Cholecystectomy was performed the next day Convalescence was entirely uneventful On 5 3 36 a follow up examination revealed that the patient was in excellent condition with no recurrence of any of the pre-operative symptoms An electrocardiogram showed an essentially normal picture (Fig 156 B)

DISCUSSION In 1908 Babcock called attention to the frequency of co-existing cardiac disease and biliary tract disease and three years later this association was further elaborated by Riesman It was the opinion of Babcock and Riesman that the cardiac disease was due to myocardial changes brought about by primary infection in the gallbladder wall In 1931 Schwartz and Hermann called attention to the fact that obesity was frequently associated with biliary tract disease and that the cardiac abnormalities might be due simply to the myocardial changes that accompany this condition More recently it has been suggested that certain of the disturbances in cardiac rhythm in patients with gall stone disease may be purely reflex in character from this focus

The frequency with which improvement in the patients cardiac function has been observed after cholecystectomy for gall stone disease the disappearance of various types of arrhythmias the improvement in the associated anginal syndrome and the return to normal of the electrocardiogram after competent biliary tract surgery has convinced many clinicians that

* Cases 102 and 103 presented and discussed by Dr I S Ravdin Professor of Research Surgery University of Pennsylvania

gall stone disease at least adds an additional load to an already handicapped heart

Fitz Hugh and Wolferth[10] several years ago called attention to the fact that patients with gall stone disease who exhibited cardiac symptoms,

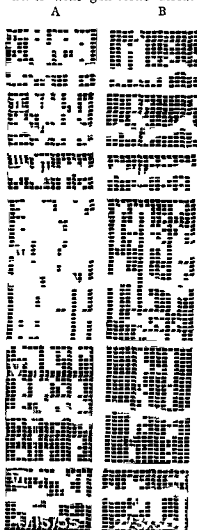



FIG 155

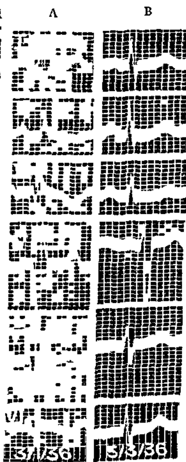



FIG 156

FIG 155 The electrocardiogram before and after cholecystectomy Note increase in amplitude of T and return to normal of the T waves in leads 4 and 5 (Old direct leads)

FIG 156 The electrocardiogram before and after cholecystectomy A change is seen in T and T of the indirect lead

often of the anginal type were greatly benefited following the removal of gall stones from the gallbladder or common duct The patients they reported were found to have abnormal electrocardiographic tracings chiefly

flat or inverted T waves in the first two leads Not only were the patients relieved of their biliary tract symptoms but these observers found that the electrocardiographic changes were reversible within a short period after the biliary tract operation These studies point to a toxic effect of the diseased gallbladder on the myocardium

It should also be remembered that gall stone disease may coexist with coronary disease in patients who have had one or more acute occlusions In a group that now numbers 68 patients who were found following complete studies to have evidences of moderate or severe myocardial disease we have been impressed with the improvement in the cardiac status following the eradication of gall stone disease It may be that the chronic gastric distention and interference with gastro-intestinal motility which so many of the biliary tract patients show plays a part in accentuating the pre-existing cardiac abnormality It has been our experience that patients with gall stone disease who also have heart disease need not be denied operation In the group of 68 patients referred to above where operations were performed for simple gall stone disease or for a stone in the common duct or both there have been but three deaths

## ILLUSTRATIVE CASES

### Abdominal Aortic Aneurysm of Arteriosclerotic Origin Simulating Acute Surgical Abdomen at Time of Dissection Prior to Final Rupture

**Case 104** F F male age 68 had been healthy except for occasional attacks of indigestion until the sudden onset of a tearing pain in the upper abdomen with radiation to the right lumbar region It was accompanied by sweating pallor and vomiting and when the patient attempted to go to the bathroom the slight exertion caused him to fall to the floor in collapse There was involuntary emptying of the bladder and bowel When first seen he was pulseless with rapid shallow respirations The picture was one of impending dissolution The abdomen showed boardlike rigidity and the temperature by rectum was 95 F In 20 minutes the patient regained consciousness and the pulse became perceptible at the wrist A diagnosis of perforated peptic ulcer was made and operation advised Further observations however changed this diagnosis when the boardlike rigidity of the abdomen disappeared A mass thought to be an aneurysmal sac was palpated On large doses of morphine and shock treatment there was rapid improvement in blood pressure color and pulse volume for 36 hours At the end of this time there was a recurrence of severe abdominal pain again accompanied by signs and symptoms of profound shock Boardlike abdominal rigidity did not reappear and the aneurysmal sac was distinctly felt It was more tender and larger in size and there was a bulging in the right flank The patient showed increasing pallor and died in coma two hours later

Autopsy showed no peptic ulcer but advanced atherosclerosis of the aorta an abdominal aortic aneurysm with rupture The entire right side of the abdomen from the spinal column to the lateral wall and from the liver to the pelvis was filled with massive retroperitoneal clot that pushed the posterior peritoneum so far forward that it was nearly in contact with that of the abdominal wall The hemorrhage had pushed into and split the mesentery of the cecum and ascending colon The mesentery of the small bowel was not affected The kidney ureter adrenal gland and other retroperitoneal structures floated in the enormous clot

RETROPERITONEAL SPACE AND STRUCTURES The aortic wall was thin and inelastic and the intima was pitted and cracked with atheromatous ulcers Just distal to th origin of the superior mesenteric artery, extending to and involving the bifurcation was a large fusiform dilatation which projected forward and to the left into th abdomen After removal this measured 14 X 8 X 6 cm The root of the mesentery lay across its anterior surface When opened the forward bulging portion was found to be filled with layers of dense y llow fibrin The wall was continuous with that of the aorta overlaid and reinforced by peritoneum and its connective tissue to which it was closely adherent The lumen of the vessel which vent through the mass against the posterior wall was lined by a smooth red layer of fresh thrombus In the central portion of this thrombus was a small fissure which overlaid a short (1 cm) irregular rupture in the posterior wall of the aorta

**Discussion** Abdominal aortic aneurysm is a rare yet important con sideration in the differential diagnosis of the acute surgical abdomen *Symptoms arising from its rupture or dissection often confuse the surgeon* in the diagnosis of abdominal emergencies puzzle the urologist when small hemorrhages invade the tissues about the kidney or press on the ureter and frequently tax the diagnostic acumen of both internist and neurologist in interpreting pain referred to various sections of the body

It is surprising how large these aneurysms may become and how great a displacement of the abdominal organs they may cause and still elude clinical detection if the sac points posteriorly and does not erode the vertebrae Thompson reported an abdominal aneurysm in a laborer age 39 which contained six and one half quarts of fluid blood and clots at autopsy The aneurysm had pushed both kidneys so far forward that they were diagnosed as metastatic masses on palpation This patient had not consulted a physician until the last few weeks of life Many cases reported in the literature were not seen until they had become moribund following perforation or dissection When subjective symptoms from these cases are tabulated, the most frequently recorded is pain It may be of any variety from a vague type of abdominal discomfort occurring at times in patients before rupture to the typical agonizing terminal variety at tending the tearing of the aortic wall The type of pain experienced by the patient in these vascular accidents is outstanding Extremely large doses of morphine seem ineffective in such calamities and this alone should suggest an abdominal vascular complication The ensuing symptoms of profound shock add further evidence Many times in the cases reported in the literature preliminary smaller ruptures may precede the final event The intermittent hemorrhages confuse the picture, and often unnecessary surgical exploration is undertaken The distribution and character of the pain at the time of rupture depend on the location of the aneurysm and *the point of rupture Extravasations into the retroperitoneal* space are frequent If either kidney or the ureter is involved renal colic is simu lated and the pain may radiate down the inner aspect of the thigh to the testicle The high site of the rupture as in this case often leads to the diagnosis of perforated peptic ulcer Vomiting and diarrhea are fre quently present and serve further to complicate the picture

Figure 157 compiled from cases reported in the literature shows the variety of symptoms that may result from rupture pressure, or dissec

tion when various structures of the body are involved (1) retroperitoneal rupture with perinephric collection simulating abscess (2) pressure on ureter with picture of uremia (3) rupture into the gastro-intestinal tract (duodenum)—pressure on duodenum gives symptoms of pyloric obstructure, (4) rupture into the peritoneal cavity (5) rupture simulating psoas abscess (6) dissection with pressure on iliac arteries followed by gangrene (7) rupture through the diaphragm into the pleural cavity with symptoms of thoracic disease (8) spinal erosion with pain

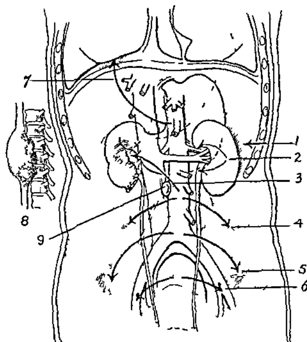



FIG 157 Diagram illustrating organs involved and pathways of dissection in reported cases of ruptured abdominal aortic aneurysm (From New Intern Clin 1 173 1939)

in the back and legs and (9) rupture into the inferior vena cava (arteriovenous aneurysm) Consequently abdominal aneurysms may produce melena hemoptysis or hematemesis Portal splenic- or mesenteric vein thrombosis may occur and give the first clue to the presence of an aneurysm

The clinical diagnosis of abdominal aortic aneurysm rests upon the demonstration of a pulsating expansile tumor mass This sign was present at the time of the second examination of Case 104 Inspection then showed a very slight anterior bulge of the abdominal wall but no pulsations could be seen At times a thrill may be palpable over the suspected mass Palpation carefully carried out usually clinches the diagnosis The tumor mass when grasped shows expansile pulsations as well as upward thrust

and it will seldom be found to move with respirations If the aneury m is high up under the diaphragm or if it points posteriorly, palpation of the sac may be difficult or impossible Often, on auscultation, a systolic murmur is heard over the sac In some instances, the patient presents himself with the chief complaint of pulsating abdominal mass (described by one patient as an "extra heart") *The pulsating aneurysmal sac must be differentiated* from the throbbing abdominal aorta and from tumor masses overlying and transmitting pulsations of the aorta In patients where the thickness of the abdominal fat is not too great and the musculature not too rigid differentiation of an aortic aneurysm from a visceral tumor may readily be made if the patient assumes the knee-chest position when the tumor will fall away from the aorta and no longer transmit a pulsation to the examining hand

It is well to remember before making the diagnosis of abdominal aortic aneurysm that a marked pulsation of the aorta is met in cases of extreme anemia in patients with aortic insufficiency in hyperthyroidism and in many underweight neurotic individuals In these cases it may be possible to grasp the vessel in the hand in which event the diagnosis of aneurysm is often made More detailed examination however will always show that only an up and down throbbing is present and no lateral expansile pulsation If the aneurysm is completely filled with clotted blood it may closely simulate a tumor mass and in these cases roentgenologic examination is invaluable Many times roentgenograms will reveal a pressure erosion in the vertebral column between the eleventh dorsal and third lumbar segments and avoid an unnecessary laminectomy

We have encountered abdominal aneurysms three times proving the diagnosis in each instance in 4 058 patients referred to the Cardiac Clinic of the Woman s College Hospital during the past ten years All were encountered in men past 60 years of age and all were of the arteriosclerotic type

For additional cases illustrating the production of abdominal symptoms by other types of circulatory disease see Cases *22* and *26*

## TRAUMA AND THE HEART

The effect of trauma on the cardiac structure and function remains a matter of much uncertainty The testimony usually heard in courts of law reflects a wide range of beliefs among physicians and shows the need of further investigations of these important problems

Penetrating wounds of the chest may involve the heart in which event the mortality rate will be high but not always 100 per cent as is the popular belief Nonpenetrating wounds are quite as capable of causing serious cardiac injury and death may follow a blow that leaves no external mark on the chest as evidence The sudden strain of lifting may at times produce cardiac injury Finally and by no means the least important of all the accident may initiate in a patient of the proper pattern

a train of symptoms of psychoneurotic nature that will center about the cardiac mechanism and greatly complicate the situation

**Importance of Cardiac Records** A complete cardiac study should be made in each case whenever cardiac injury is suspected even though a psychoneurotic factor may be evident from the start These records will at least give the examiner something to work on All the abnormalities in the electrocardiogram and the roentgenogram should be studied and carefully listed Of course the question always arises in court as to the presence before the accident of the changes described It is quite possible for heart disease to be present in some patients who may have had no symptoms related to the circulatory apparatus and no antecedent history of disease likely to produce cardiac damage While the increasing efficiency of the medical supervision of all industrial workers particularly those engaged in the dangerous occupations many times affords the opportunity for this comparison yet in the great majority of instances when the question arises as to the origin of some defect of structure or function previous studies are not available Physicians in private practice usually keep poor records and this occasionally deprives a patient who has been in an accident of the opportunity of receiving a just decision when the claim for damages is based on cardiac trauma It is also possible in cases where the heart has been damaged before the accident that a greater degree of traumatic injury may result Records showing the previous cardiac status give an opportunity for comparison especially in cases where an electrocardiogram and orthodiagram have been made before the accident and an excellent chance for evaluation of post traumatic symptoms and signs

When we consider the difficulties that surround the study of many other organs of the body we must confess that cardiac examination is easy The heart can be viewed by means of the fluoroscope against the clear pulmonary background and its mechanisms can be studied accurately by the electrocardiograph Consequently if we always consider the possibility of cardiac lesions following trauma of any type and carefully record all departures from normal the nature and extent of these lesions in time will be much better understood

**Relation to Pre Existing Disease** Trauma may accentuate any type of pre existing cardiac disease For example slight degrees of trauma may be sufficient in cases of coronary arteriosclerosis complicated by occlusion and infarction to cause rupture of the heart It is entirely possible for trauma to be a factor in precipitating attacks of congestive failure in a patient with a delicately balanced circulation Anginal seizures may be brought on in one already subject to them by the nervous upset that accompanies an accident and this attack may be the one that results fatally Among workmen who hold dangerous jobs the sudden onset of angina paroxysmal disorders of rhythm or attacks of cardiac syncope may cause fatal accidents There is much disagreement concerning the likelihood of trauma predisposing to an attack of acute

rheumatic fever or to a blood stream invasion by an organism like the *Streptococcus viridans* with the production of subacute bacterial endocarditis However, we cannot deny the fact that, in a patient who has subacute bacterial endocarditis, even moderate trauma may cause the rupture of a previously damaged heart valve

## PENETRATING WOUNDS

The most common form of cardiac trauma is that produced by knives bullets or fragments from fractured ribs or sternum These cases are surgical if they survive long enough to be brought to the hospital While many penetrating wounds are immediately fatal a small percentage of cases may be saved by prompt recognition and suture Since no time can be lost when the emergency arises every surgical service should have established rules for diagnosis and treatment of these injuries Elkin[91] states that approximately 2 per cent of the penetrating wounds of the chest involve the heart If all cases were considered the figure would no doubt be higher, since this statement is based on cases that survived long enough to reach the hospital In Elkin s series of 22 cases wounds of the heart were produced either with a knife (15 cases) or ice pick (seven cases) Bullet and gunshot wounds are rarely met in hospital practice since death from hemorrhage usually takes place before emergency measures can be applied

### SYMPTOMS

The patient who has just received a stab wound of the heart usually is unconscious when first seen In some instances delirium may be present The skin is cold the lips show a pallid cyanosis the heart sounds are weak and distant and the pulse is usually absent These symptoms are caused by loss of blood and beginning tamponade of the heart Free bleeding into the pericardial sac follows the wound in the heart wall When a sufficient quantity of blood collects cardiac action is embarrassed diastolic relaxation is incomplete and the venae cavae are unable to empty completely into the right auricle Consequently an increase in the venous pressure takes place which is evident clinically in the distended condition of the veins of the neck and the cyanosis of lips and tongue As the venous pressure increases the arterial pressure drops, and usually blood pressure readings are soon unobtainable If a portable bedside fluoroscope is handy increase in heart size may be seen with an absence of normal pulsations along the cardiac border produced by the accumulation of blood in the pericardial sac If these symptoms of tamponade are demonstrable following a stab wound cardiac suture should be attempted at once Although the patient is unconscious at the beginning of the procedure relief of the tamponade will increase blood pressure decrease venous pressure and re establish cerebral circulation with a quick return of consciousness Consequently, although the patient is comatose at the start an anesthetic should always be used

### PROGNOSIS

The prognosis depends on the speed with which the condition is recognized and operation performed If immediate death from hemorrhage does not occur cardiac tamponade may be quickly fatal while after operation purulent pericarditis and pneumonia often prove serious complications The location of the wound is significant from the standpoint of prognosis In Elkin s series six out of eight patients who had wounds of the right ventricle recovered Left ventricular wounds are more serious only two in this location recovering in the series of 22 cases reported by Elkin Wounds of the pulmonary artery the auricles and intrapericardial part of the aorta have a poorer prognosis because of the technical difficulties involved in successful suture

Rarely some foreign bodies lodge in the heart wall and become encysted others do not remain in one location but migrate until they are free in one of the cardiac chambers in which event embolism may take place The great veins in some reported cases have conveyed foreign bodies to the heart from distant wounds

## Non Penetrating Wounds

Equally serious cardiac injuries can be produced by contusion or compression Beck calls attention to our erroneous conception of the heart as an organ that is well protected by a long thoracic cage whereas in truth it lies against the sternum vulnerable to any sudden blow on the chest and capable of being buttressed against the bodies of the thoracic vertebrae posteriorly

**Penetration of Myocardium** Blows over the precordium may fracture the sternum or ribs and the cavity of the heart may be penetrated by the ends of the fragments A partial penetration of the muscle of the heart may result in healing with subsequent cardiac rupture or the formation of a cardiac aneurysm A cardiac bruise from this type of injury may also heal with the production of subsequent functional disturbances Sudden indirect forces resulting from falls may cause compression of the legs on the abdomen and injure the heart while violent blows on the chest may cause laceration of a cardiac chamber or the aorta and result in sudden death with no external evidence of injury

**In cardiac rupture** the cause of death is neither the injury nor the hemorrhage it usually is cardiac tamponade In a certain number of these cases where the outcome is not immediately fatal careful examination will reveal the tamponade in which event prompt action saves the patient s life RECOGNITION OF THE PRESENCE OF ACUTE TAMPONADE SHOULD NOT BE DIFFICULT IF THE POSSIBILITY OF ITS OCCURRENCE IS ALWAYS KEPT IN MIND WHEN EXAMINING AN ACCIDENT CASE As an emergency measure Beck[4] advises tapping of the pericardial cavity at intervals until operation can be performed This procedure (page 164) serves to confirm the diagnosis and to relieve temporarily the cardiac tamponade

A few months ago I observed one case where the seriousness of the signs of cardiac tamponade were unrecognized following a stab wound of the heart with an ice pick in the fifth interspace about 8 cm from the midsternal line The patient was delirious on admission to the hospital and later pulseless Spontaneous recovery took place without operative interference Such occurrences, however, are rare

SYMPTOMS

**Immediate Onset** Cardiac trauma from non penetrating wounds of the chest may cause symptoms closely resembling those that follow a coronary occlusion If a patient previously in good health receives a blow over the precordium and immediately thereafter develops dyspnea irregularity of the pulse and pain in the chest of the anginal type cardiac trauma should always be considered These symptoms often lead to a diagnosis of cardiac neurosis with which trauma is so often confused If accompanying these symptoms electrocardiographic changes appear that were not observed prior to the accident the presence of a traumatic lesion becomes more than a possibility

*Late Onset In some cases the onset of the symptoms* does not immediately follow the accident instead, the symptoms appear at varying intervals depending on the size location and the progress of the myocardial lesion The latter may result in scar formation but the patient is always liable to develop cardiac aneurysm or cardiac rupture at a later date, particularly if rest is not enforced Contusions of the heart which are productive of large areas of myocardial injury, therefore may in some instances behave quite like cardiac infarcts

**Functional Disturbances** Cardiac trauma may be followed by disturbances of a functional nature including arrhythmias angina and congestive failure

Congestive Failure Either right or left ventricular failure may appear on effort during the post traumatic period, particularly in the presence of an already damaged heart The onset may follow trauma immediately or it may be delayed for some time if the lesion produced in the cardiac structure is of the type that slowly reduces myocardial reserve for example rupture of a valve or prolonged paroxysms of abnormal rhythms accompanied by high ventricular rates Beck's experiments point to the fact that severe contusion of a perfectly healthy heart may at times lead to acute dilatation and decompensation

Angina While trauma directly applied to the coronary tree may occasionally provoke symptoms of angina in a normal person the likelihood is even greater if coronary arteriosclerosis is present The nervous shock that results directly from the accident may reflexly cause coronary spasm and in a susceptible person the anginal seizure may appear If chest pain is complained of immediately after trauma and is followed by the sudden death of the patient the effect of the accident on the integrity of the coronary circulation may be strongly suspected Characteristic alterations

in the T waves of the electrocardiogram appearing after the accident lends support to the diagnosis

ARRHYTHMIAS

The appearance of arrhythmias following trauma many times focuses attention on the heart Premature beats or extrasystoles may be noticed for

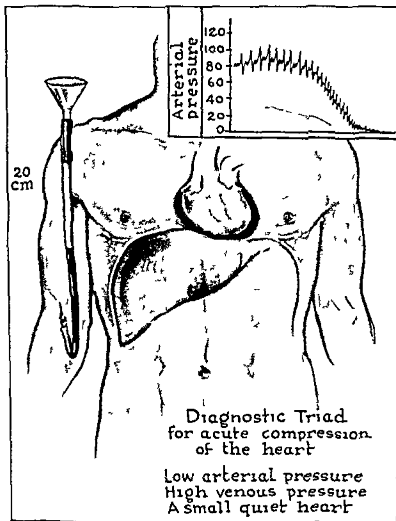



FIG 158 Acute compression of the heart Redrawn from Beck *

the first time after an accident and the more attention is directed toward them the more important they seem to the patient In many instances

* Reprinted by the courtesy of the editor of the American Heart Journal

cardia may be the direct result of myocardial injury and should be promptly controlled because of the tendency of this arrhythmia to terminate in ventricular fibrillation and death

Cardiac injury may bring on an attack of auricular flutter or fibrillation in either normal or diseased hearts If the heart is healthy and not extensively involved in the trauma the abnormal rhythm is usually easily controlled However if the heart is damaged and the arrhythmia is continuous with a high ventricular rate congestive failure may be precipitated

HEART BLOCK Injury to the heart muscle that is followed by hemorrhage in the region of the conduction system may cause varying degrees of heart block Similar lesions have been reported in experimental animals and some case reports in literature point to this occurrence in man To establish this happening beyond a doubt it is necessary to have in evidence a normal electrocardiogram taken before the accident and an electrocardiogram showing the appearance of heart block directly following the trauma

## ILLUSTRATIVE CASE

### STAB WOUND OF THE RIGHT VENTRICLE—ACUTE CARDIAC TAMPONADE—RECOVERY FOLLOWING SUTURE

**Case 105** A E an American school boy of 14 was brought to the receiving ward of Memorial Hospital on December 8 1938 by police ambulance approximately one half hour following a stab wound in the left chest with a pocket knife

PHYSICAL EXAMINATION The patient was unconscious on arrival and showed a pale cyanosis The jugular veins were distended The pulse was imperceptible at the wrist and the respirations were rapid and shallow The heart sounds were faint the rhythm regular and the cardiac rate was 70 The blood pressure could not be obtained by ordinary clinical methods There was a stab wound one half inch long in the fifth intercostal space immediately to the left of the sternum Active bleeding had ceased The liver was not palpable The extremities were cold and pale

DIAGNOSIS Acute cardiac compression (Fig 158)

INDICATION Immediate operation

OPERATION (Dr Bruce Fleming) As anesthesia was begun incision was made parallel to the ribs and the wound of entrance completely excised down to the pleura The incision was enlarged and four inches of ribs above and below the incision were removed The pericardial sac was greatly distended contained a puncture wound and was completely filled with a firm blood clot The thoracic cavity contained blood and clots The pericardium was incised through the wound and clots removed Traction sutures of silk were placed in the myocardium, one near the apex centrally and one cephalad to the wound in the heart while bleeding from the latter was controlled by the finger of the assistant The estimated size of the myocardial puncture was three eighths of an inch Two through and through chromic gut sutures were placed in the myocardium completely closing the wound Blood was removed from the thorax and pericardium and strained into citrate solution and returned to the patient intravenously The control sutures were then removed and the pericardium sutured The thoracic wall was closed without drainage Dressings were applied

COURSE Following operation the patient was placed in an oxygen tent The pulse became palpable as soon as the pericardial sac was opened and the acute cardiac compression relieved At this time the first blood pressure reading obtained was 98/56

A roentgen ray examination mad on the first postoperative day (Fig 159 B) showed a uniform density over th left hemithorax more marked at the base which obscured the lung d tail The heart and mediastinum were d splaced to the right Conclu on Pleural effusion with card ac displacement

An examination made on the fifteenth postoperative day showed a decrease in the effusion The pleural was th ckened and the heart wa still d splaced to the right

A roentgen ray exam nation two months later showed that the left lung had completely re-expanded with absorption of the fluid at the left bas The h art and m diast num occupied normal posit ons

The blood count immediately following operation was hemoglobin 50 per cent R B C 900 000 W B C 14 000 P 8 L 18 Following s veral tran fus ons it was raised to hemoglobin 8 per cent R B C 4 460 000

Electrocard ograms were taken at frequent intervals postoperatively Th chang s that took place with healing are shown in Fig 159 C

The patient mad a good recov ry and was discharg d from th hospital s ven w ks after the ope ation

# 18

# CARDIAC EMERGENCIES

While heart disease in most instances gives ample warning of its presence often assuming the form of a chronic illness, situations may arise that demand quick decision and prompt treatment Although these have been discussed fully under their appropriate headings elsewhere in the text, for convenience and quick reference I have attempted to group here the cardiac emergencies commonly encountered with some of the present day views regarding their diagnosis and treatment

Cardiac emergencies are common in surgical as well as in medical practice Many times treatment must be begun before a complete physical examination is made Likewise in some instances the past history is unknown or difficult to obtain in detail Since the leading symptoms must be relieved in some measure before the etiologic possibilities are reviewed it seems appropriate to begin a discussion of emergency treatment with a review of the symptoms of cardiovascular origin that are usually responsible for these calls These are chest pain, dyspnea syncope and palpitation

## CHEST PAIN

Chest pain in cardiac patients requiring emergency treatment may be caused by acute coronary occlusion angina pectoris, pulmonary embolism acute pericarditis, or dissecting aneurysm Angina pectoris and coronary occlusion the complications of arteriosclerotic heart disease are the most common causes The significant features in their differentiation that may prove useful in emergencies are summarized in Table XIV

**Coronary occlusion** is the most frequent cardiac emergency associated with prolonged chest pain and the increasing familiarity of the layman with the symptoms of this condition usually bring minor episodes promptly under the supervision of the physician The pain of occlusion calls for immediate administration of morphine in sufficient doses to quiet the patient It is best to give an initial dose of 15 mg (1/4 grain) and if necessary, repeat the dose in 15 to 20 minutes Quick relief of pain is a major factor in turning the balance in favor of recovery Do not give digitalis unless congestive failure is precipitated If the patient is at home he should be made comfortable in the room where he is found until his condition permits his removal If the seizure takes place on the street the patient should be moved to the nearest shelter to await transportation to the hospital Oxygen therapy if available is an advantage particularly in the presence of cyanosis Injection of concentrated glucose (50 Gm in

TABLE XIV

DIFFERENTIAL DIAGNOSIS IN CHEST PAIN

| | ACUTE CORONARY OCCLUSION | ANGINA PECTORIS |
|---|---|---|
| Appearance of patient | Restless ashen color dyspnea sweating Shock may be present Congestive failure may develop | Patient quiet and immobile Good color No dyspnea No sweating |
| Pulse | Rapid and weak Paroxysmal ventricular tachycardia present occasionally | Normal |
| Blood Pressure | Usually falls to low level Pul us alternans may be observed (page 407) | Normal or elevated |
| Temperature | Subnormal when first seen | Normal |
| Heart Examination | Heart sounds weak and often rapid Gallop rhythm is not uncommon Friction may be audible if the area of infarction is on the anterior surface | May be normal |
| *Duration of pain* | *Hours* | *Few minutes* |
| Onset | At any time Common during sleep | Following exertion excitement or overeating |
| Effect of nitrites | Pain not relieved This drug may be harmful | Quick relief usual |

100 cc ) into the arm vein may give quick relief to a failing myocardium Coronary occlusion rarely occurs without pain When it does, other symptoms of collapse of the circulation may appear suddenly especially severe dyspnea (cardiac asthma) The onset of an attack of paroxysmal dyspnea particularly when a previous history of anginal attacks is elicited should always suggest the diagnosis of acute coronary occlusion

**Other Conditions** Herrick[1] has called attention to a number of other conditions that may be associated with chest pain and consequently be confused with acute coronary occlusion He lists under this category effort syndrome cardiac neurosis cardiac arrhythmias pericarditis syphilitic aortitis aortic aneurysm pleurisy pneumonia massive collapse of the lung bronchial carcinoma pulmonary embolism *herpes zoster spinal* osteo arthritis gall stones peptic ulcer neuralgic or fibrotic causes local to the chest wall hernias through the diaphragm carcinoma of the stomach and tabetic crises

If the chest pain is transient and the patient when first seen by the physician has a good pulse and blood *pressure and on auscultation the* heart sounds are unchanged angina pectoris rather than occlusion may be suspected A tablet of nitroglycerine 0 6 mg (1/100 grain) under the tongue should bring prompt relief If the pain continues a similar dose may be given in five minutes After emergency relief has been obtained the patient should be advised to have *a complete cardiac study* made in the near future

**A large embolus** lodging in one of the branches of the pulmonary artery may obstruct the flow of blood from the right side of the heart (acute cor pulmonale) and cause sudden death If the patient is alive when

seen in advanced degree of shock is present The symptoms may then resemble acute coronary occlusion chest pain pallor, thready pulse dyspnea, falling blood pressure and shock The differential diagnosis in some cases may be most difficult if not impossible to make However, the points favoring pulmonary embolism are a recent surgical operation (abdomen or pelvis) fracture, phlebitis, or chronic cardiac disease where an auricular thrombus may have formed An acute cor pulmonale may produce a pulsation in the second or third interspaces just to the left of the sternum occasionally accompanied by a friction rub dilated jugular veins and marked cyanosis Many times these signs are lacking If the patient survives long enough for an electrocardiogram to be made this may help to make the differential diagnosis (see Fig 251) Later cough hemoptysis, and signs of consolidation over the area of the pulmonary infarct may serve to clear the picture

Absolute bed rest morphine oxygen and support of the failing right heart by venesection and if the emergency is extreme intravenous injection of strophanthin comprise measures of value in cases of pulmonary embolism Surgical removal of the embolus is occasionally possible when the accident occurs in the hospital in the presence of a skilled surgical team

**Acute pericarditis** may cause sudden chest pain in younger patients but this is a rare happening The pain usually is caused by the extension of the inflammation to the pleura and diaphragm The age of the patient, and evidences of rheumatic infection serve to differentiate this type of pain from that caused by anoxemia of the heart muscle that occurs in coronary disease

## DYSPNEA

A sudden attack of dyspnea (usually nocturnal) may be the first symptom of heart disease to appear It is the most common cardiac emergency of general practice The patient usually a man is awakened suddenly by a sense of suffocation Dyspnea is present and it quickly increases to orthopnea When the physician arrives he finds the patient's chest full of rales the color ashen and the general appearance one of impending death There may be cough with frothy blood tinged sputum This is the picture of cardiac asthma or paroxysmal cardiac dyspnea

**Immediate Treatment** Unless the sudden left ventricular failure is promptly relieved it may terminate in acute edema of the lungs Morphine, 15 mg (¼ grain) given hypodermically and repeated if necessary in 20 minutes is usually most efficient in bringing the attack to an end When pulmonary edema complicates the picture a venesection should be done although venostasis or bloodless venesection may prove helpful The latter procedure consists in applying a tight bandage to the arms and upper thighs close to the trunk to prevent the return of venous blood and in this manner diminish the pulmonary congestion When the attack

subsides the compression bandages are gradually released If the patient is in the hospital when the attack occurs an oxygen tent is useful

When the emergency treatment has been given further observation of the patient who is recovering from a sudden attack of cardiac asthma will usually disclose other evidences of heart disease The victims of these attacks almost always suffer from the effects of a long standing hyper tension and show cardiac hypertrophy chiefly left ventricular In many instances the gallop rhythm of failure may be heard on auscultation in the region of the cardiac apex Pulsus alternans may also appear in these cases If the examination reveals any of these signs a guarded prognosis should be given to the patient s family The duration of life of older patients who have attacks of cardiac asthma is hardly ever over one year from the time of the first attack

**Careful follow up treatment** is recommended in all cases seen in the first attack of cardiac asthma Digitalization should be carried out (page 82) and suitable doses of diuretic drugs particularly the organic mercurial group should be given even in the absence of edema for the effect they often have in preventing the recurrence of the seizures The patient should be told to sleep in a semi reclining position and to avoid slipping down in the bed during the night

**Allergic or Bronchial Asthma** Spells of dyspnea caused by allergic or bronchial asthma in an elderly patient may be confusing at times (page 114) The presence of hypertension and the cardiac findings above described point to a cardiac background particularly if the attacks are of recent origin A history of chronic cough wheezing throughout the day attacks of asthma for many years the presence of emphysema and a recent upper respiratory infection point to bronchial asthma Epinephrine is indicated in the bronchial or allergic type of asthma but is strongly contraindicated in cardiac asthma (Chapter 16)

## SYNCOPE

When the cause of syncope is cardiac the physician obtains his first diagnostic clue from the rate and rhythm of the pulse When no pulse is felt at the wrist or when it is under 20 beats per minute in an elderly person an Adams Stokes seizure should be suspected Since the auricles continue to beat evidence of their activity should be looked for in the neck veins If regular pulsations can be demonstrated proof of the diagnosis is furnished (page 375) In the presence of an Adams Stokes seizure intracardiac injections of epinephrine should not be used indiscriminately when the exact cardiac mechanism present during the seizure is unknown Where ventricular tachycardia characterizes the attacks the careless use of epinephrine may remove the only chance the patient has of spontaneous recovery (page 403)

**Paroxysmal Tachycardia** The sudden onset of paroxysmal rapid heart action may in some instances be accompanied by a marked diminu

tion in the supply of blood to the brain Syncope follows If an attack of auricular flutter is accompanied by one to one ventricular response the unusually rapid heart rate may be incompatible with circulatory efficiency Paroxysmal tachycardia may likewise produce syncope in elderly patients where advanced sclerosis limits the cerebral blood flow In desperate situ ations if the patient has not been taking digitalis previously and the paroxysms are accompanied by the sudden onset of congestive failure intra venous use of strophanthin, 0 6 mg (1/100 grain) is justified (page 398)

In paroxysms of tachycardia in young people when no signs of cardiac embarrassment are in evidence less heroic measures suffice (ice water aromatic spirits of ammonia, whiskey taken straight in tablespoonful doses carotid sinus pressure quinidine eye ball pressure, holding the breath bending over with the head between the knees, heat or cold over the precordium) When the patient s condition is good there is no oc casion for alarm for the paroxysms of tachycardia are self limiting Re cumbency, however should be maintained during the seizure to prevent vertigo or syncope

**Carotid Sinus Syncope** Fainting attacks often accompany hyper sensitiveness of the carotid sinus and these may be associated with a slow pulse Recovery is prompt and the diagnosis of the nature of the attack usually offers little difficulty (page 378)

**Emotional Syncope** Certain types of patients are always fainting and sending for the doctor Emotional episodes in the presence of neurocircu latory instability precipitate the attack of syncope A history of previous seizures the good quality of the pulse and the absence of any signs of heart disease on examination suffice to make the diagnosis

**A high vagal tone** associated with sinus arrest may produce syncope (page 379) The treatment is recumbency and atropine

**Aortic Lesions** At times patients with aortic stenosis and aortic regurgitation are subject to spells of syncope Harrison[149] attributes these to an anoxemia that is diffuse involving the left ventricle at the time of increased activity Syncopal attacks occasionally observed in children with patency of the ductus arteriosus may be caused by a similar mechanism since the mechanical effects of this defect are quite like those produced by an aortic regurgitant lesion

**Extracardiac Factors** Syncope is produced many times by factors un related to the heart The more common types I shall mention here, since in an emergency a differential diagnosis from heart disease is required Epilepsy can be differentiated from the Adams Stokes seizures on the basis of the tongue biting the character of the convulsive movements and the quality and rate of the pulse during seizures In cerebral acci dents the type of breathing and the evidence of paralysis are dis tinguishing features Sudden hemorrhage may be followed by syncope and this may be accompanied by a rapidly rising pulse and marked pallor

**Thrombi** In mitral stenosis and following a coronary occlusion mural thrombi may form in the heart and these may be swept into the circulation at any time with the sudden appearance of symptoms the nature of which will depend on the distant area affected Vegetations in subacute bacterial endocarditis may likewise become detached with similar sequelae The sudden onset of coma and paralysis should always suggest these complications to the physician who is summoned in the emergency

## PALPITATION

The sudden appearance of palpitation often accompanies one of the abnormal rhythms Initial attacks of paroxysmal tachycardia may cause great alarm on the part of the patient and his family while emergency medical aid is less likely to be sought for the subsequent attacks unless they are unusually severe or prolonged Abnormal rhythms should be differentiated from simple tachycardia The latter is usually present in patients showing other signs that label the episode as purely functional The heart rate while rapid will be noted to decrease gradually Such is not the case in patients suffering from attacks of paroxysmal tachycardia for in these both onset and offset are sudden

**Abnormal rhythms** commonly encountered are auricular fibrillation auricular flutter and paroxysmal auricular or nodal tachycardia Since these mechanisms have already been fully discussed (Chapter 12) only a summary of important data will be included here

Auricular fibrillation when paroxysmal may occasionally be observed in the absence of any other sign of heart disease It frequently accompanies hyperthyroidism If the patient is in good condition and the heart otherwise normal the only therapeutic measures indicated are rest and the administration of a capsule of quinidine sulfate 0 3 Gm (5 grains) If cardiac disease is present and the heart is increased in size particularly if signs of congestive failure are recognized QUINIDINE SHOULD NEVER BE GIVEN If the patient has not previously been receiving digitalis and this fact can be definitely established there is no contraindication to the intravenous administration of strophanthin if the abnormal rhythm is placing a severe strain on the myocardium and early signs of cardiac failure are appearing Enough digitalis should be given subsequently by mouth to maintain the ventricular rate at 70 to 75

A persistently rapid and regular cardiac rate between 130 and 160 beats per minute is more apt to be flutter than tachycardia The breathing the color of the patient and the presence of any signs of cardiac failure should guide the physician in his estimation of the condition of the myocardium and provide a key to the type of emergency treatment to use Here again if evidence is at hand that digitalis has not been previously given full doses may be prescribed to control the ventricular rate The future course of therapy depends on subsequent studies (page 393)

Paroxysmal tachycardia is a common cause of the sudden onset of palpitation in patients who have no other evidence of heart disease The

prognosis in young individuals even in the presence of extremely rapid ventricular rates (160 to 200) is invariably good and death in a paroxysm is practically unknown Normal rhythm may reappear before the physician arrives If so there remain only the effects of the seizure on the nervous system to treat in which event a few doses of phenobarbital suffice If the attack persists the measures previously described may be instituted in order (page 506) A follow up study is needed to determine the underlying cause and to establish, if possible, satisfactory prophylactic treatment (page 382)

## WOUNDS OF THE HEART

Puncture wounds of the heart may be small with the gradual development of a cardiac tamponade by leakage of the blood into the pericardial sac Since many of these cases can be saved by emergency surgical measures (page 494) it is important to act promptly in the presence of the following signs fall in blood pressure rise in venous pressure dyspnea progressing to orthopnea cyanosis tachycardia and decreasing pulse volume They are all produced by a rising intrapericardial pressure compressing the heart and preventing proper diastolic filling

## PERICARDIAL EFFUSION

Occasionally as a pericardial effusion slowly accumulates the symptoms of cardiac tamponade may appear at a much slower rate Nevertheless when the intrapericardial pressure becomes elevated the situation becomes an emergency and prompt treatment may be life saving The technic for aspiration of the pericardial sac is outlined on page 164

## CONGESTIVE HEART FAILURE

In a small number of cases the symptoms of congestive cardiac failure come on suddenly and emergency treatment is needed If digitalis is given under these circumstances to the patient of another physician the amount is gauged by the previous dosage (page 82) If this cannot be determined it is wise to proceed with caution Bed rest and morphine are safer measures to employ Severe dyspnea may be caused by large pleural collections of fluid in which event a thoracentesis may be useful as an emergency measure (page 94)

## CARDIAC RESUSCITATION

Ventricular standstill or ventricular fibrillation are the mechanisms usually responsible for sudden death When either occurs in a badly damaged heart all attempts to restore co-ordinated contraction are futile However as Beck has shown a normal heart that stops suddenly can

be made to beat again This accident may occur during operations as a result of the toxic effect of the anesthetic hemorrhage or cardiac trauma or it may be produced reflexly by careless handling of the abdominal viscera

Defibrillation of the ventricles has been accomplished by Beck His method is described on page 474 If ventricular standstill is present intra cardiac injection of epinephrine may be successful (page 475)

In emergencies when efficient cardiac contractions suddenly cease the first thought should be to maintain the blood supply to the vital cerebral structures Oxygen should be given through a face mask and direct cardiac massage begun to prevent irreparable damage in the brain areas while measures for restoring normal cardiac contractions are being instituted

## ACUTE CORONARY OCCLUSION

### Symptoms and Signs

1 **Pain** Usually lasts an hour or more but in rare cases it may be absent An attack of paroxysmal dyspnea or cardiac asthma may be the presenting symptom of the occlusion
2 **Shock** Patient usually cold, pale, sweating with rapid, thready pulse and low blood pressure In small occlusions shock symptoms may be be very few or absent altogether
3 **Fever** leukocytosis friction rub, acceleration of the sedimentation time are LATER SIGNS

### Treatment

Rule If the patient presents the textbook picture, the diagnosis is easy If in doubt, be cautious and prove the absence of occlusion before allowing the patient to be up and about

1 **Bed Rest**
2 **Morphine for Pain** Give enough to control the pain satisfactorily usually 0 016 Gm (¼ grain) at first dose and repeat in half hour if necessary and every two or three hours thereafter as required Respiratory rate is guide to dosage Nitroglycerine is useless
3 **Oxygen** A great advantage if available Use nasal catheter method in the home (page 99) A tent (page 101) is the method of choice in the hospital Oxygen should be continued until the pain disappears and the cyanosis is absent
4 **Digitalis is Contraindicated in Acute Coronary Occlusion Unless Symptoms of Cardiac Failure are Precipitated by the Accident** Digitalis may cause an attack of ventricular fibrillation Quinidine has been recommended for routine administration to prevent this complication (page 274)
5 **Glucose Is of Value** It increases available glycogen and provides food for the distressed myocardium Give 50 cc of 50 per cent solution very slowly into the vein using a 50 cc syringe in an emergency
6 **For Shock** caffein sodium benzoate 0 5 Gm (7½ grains) hypodermically

# HEART FAILURE

## I CONGESTIVE TYPE
(page 73)

### SYMPTOMS

Dyspnea progressing to orthopnea Edema Large liver Rales in the chest Increased venous pressure Any arrhythmia may be associated with congestive failure

### TREATMENT
(page 74)

1 **Bed Rest**
2 **Morphine** 016 Gm (¼ grain) third hour as required
3 Obtain an experienced attendant at home or send the patient to the hospital
4 **Restrict Fluids**
5 **Digitalization** (page 75)
6 **Mercurial diuretics** (page 88)
7 **Oxygen** (page 98)

## II PAROXYSMAL CARDIAC DYSPNEA OR CARDIAC ASTHMA
(Acute left ventricular failure)

### SYMPTOMS

Those of advanced heart disease Sudden attacks of dyspnea occur usually at night and may progress to orthopnea The lungs quickly fill with rales Later edema occurs Death may follow in the absence of prompt treatment

### TREATMENT

1 **Morphine** 15 mg (¼ grain) by hypodermic immediately
2 **Venesection** 400 to 600 cc (Indications cyanosis distended jugular veins and dyspnea)
3 **Oxygen** if available Nasal catheter method at home Tent if the patient is in the hospital
4 **Aminophyllin** 0 24 Gm (3 to 4 grains) intravenously
5 If attacks recur frequently mercurial diuretics may be valuable in lessening the incidence in some cases

## THE PAROXYSMAL TACHYCARDIAS

### Auricular

Heart rate is 160 to 200
Onset sudden Offset sudden

#### SYMPTOMS

**Palpitation**
**Vertigo**
**Syncope (rare)**

#### DURATION

*Few minutes to few hours*
*Very rarely paroxysms continue over a week*

#### CAUSE

None may be present
Heart usually normal
Heart disease may be present (most common type rheumatic)

#### TREATMENT

Try in order
- **Carotid sinus pressure** (page 379)
- **Vagal stimulation**
  - **Eye ball pressure**
  - **Tickle throat to cause gagging**
  - **Emetic (ipecac)**
- **Quinidine** (page 383)
- **Mecholyl (?)**

### Ventricular

Heart rate rapid (120 to 160) regular At times may be irregular

#### SYMPTOMS

Onset abrupt Premature beats occur before seizure occasionally in runs of two or three Often these may be recognized and act as a warning
Heart disease present usually serious Commonly coronary occlusion

#### TREATMENT

**Quinidine**
**Carotid pressure *ineffectual***
**Digitalis dangerous**

## AURICULAR FLUTTER

1 Heart rate 100 to 150 It may change
2 Onset is sudden
3 The termination may be gradual or sudden
4 Heart disease is usually present
5 Carotid sinus pressure often slows the ventricular rate

### Treatment

Digitalize This changes flutter to fibrillation Withdrawal of the digitalis may then cause the rhythm to return to normal If advanced heart disease is present a maintenance dose of digitalis should be continued (page 394)

Quinidine has been used to stop paroxysms if no heart disease is present or cardiac damage is slight Digitalis however is the drug of choice

## AURICULAR FIBRILLATION

1 Heart rate 100 to 160
2 Rhythm Irregular EXERCISE INCREASES THE IRREGULARITY
3 Heart disease
   Almost always present
   In rare instances the heart may be normal
4 The onset is sudden
5 Termination may be abrupt

### Treatment

Digitalize if heart disease is present (page 395) A good response to the drug may be predicted In instances in which fibrillation is paroxysmal and heart damage is slight quinidine is indicated

## ADAMS STOKES SEIZURES

### Symptoms

Syncope Convulsive movements No paralysis Evidence of arteriosclerosis is apt to be present and the patient is usually beyond middle life There may be a previous history of seizures The pulse is slow (10 to 30 per minute) or the patient may be pulseless Recovery occurs in a few minutes to five minutes Death may occur in any of the seizures

### Mechanism

There may be a cardiac standstill or prefibrillary type of ventricular tachycardia with short runs of ventricular fibrillation These cause cerebral anemia

### Heart Disease

This is present usually with partial A V block alternating with periods of complete A V block Normal sinus rhythm is seen in rare cases The heart block usually follows a gradual or acute occlusion of a coronary artery Other causes of heart block (gumma tumor, endocarditis etc ) are rare

### Treatment

Usually unsatisfactory at the time of the seizure If electrocardiographic proof of cardiac standstill has been obtained intracardiac injection of epinephrine may be given (page 475) Use 0 5 cc of a 1–1000 solution In an extreme emergency do not wait to boil up equipment Use lumbar puncture needle and 2 cc syringe LIMIT USE OF INTRACARDIAC INJECTIONS TO THIS TYPE OF EMERGENCY AND CASES OF SUDDEN CARDIAC STANDSTILL DURING ANESTHESIA

### Follow up Advice

Bed rest until a complete cardiac study is obtained To prevent recurrence of attacks many drugs have been recommended These include ephedrine benzedrine thyroid extract, metrazol and barium chloride (page 403)

## EMBOLISM

### Mechanism

**Detached thrombi** (1) from auricles in cases of mitral stenosis or subacute bacterial endocarditis and from ventricles in cases of coronary occlusion or subacute bacterial endocarditis (2) from pelvic or thigh veins on tenth to fourteenth postoperative day Emboli go to lungs or systemic vessels depending on the point of origin From right heart destination is lung except in rare instances where there is a patent foramen ovale Air or fat emboli follow trauma

### Symptoms

If embolus lodges in extremity there is first PAIN followed in a few hours by pallor and drop in temperature on affected side No arterial pulsation is present below the point of occlusion

### Treatment

1 Morphine for pain
2 Keep the patient warm and combat shock The extremity should be wrapped in cotton
3 Oxygen for pulmonary embolism in presence of cyanosis and dyspnea (page 98)
4 Consider embolectomy if the site is known and the vessel can be quickly reached by the surgeon If gangrene present amputation
5 **Avoid massage**

### Preventive Measures

These are important postoperatively in the prevention of pulmonary embolism (acute cor pulmonale)
1 Trendelenburg position
2 Carbon dioxide administration several times daily during the first 48 hours
3 Frequent deep breathing exercises
4 Keep extremities warm both during and after operation
5 Massage and passive motion of legs during the first 48 hours after operation
6 Thyroid gland (U S P ) may speed venous return and is useful in carefully selected cases

## PERICARDIAL EFFUSION

### Etiology

Pneumonia In this disease the effusion is apt to become purulent (page 171)
Tuberculosis Large effusions often occur (page 177)
Rheumatic fever Large effusions are rare but can occur (page 162)
Malignancy Large effusions rare

### Signs

Friction rub
Fever
If fluid accumulates in amounts above 300 cc, signs of compression of the the heart may develop The onset of symptoms may be sudden dyspnea, cyanosis increased venous pressure and fall in pulse pressure (page 164)

### Treatment

Pericardial paracentesis For technic see page 166
Surgical drainage if effusion purulent (page 174)
Sodium cacodylate intravenously in daily doses of 5 to 8 grains (Willius)

## WOUNDS OF THE HEART

### Signs

Shock with evidence of puncture wound of the chest in cardiac area Falling blood pressure increase in venous pressure and other signs (if patient survives) of acute cardiac tamponade

### Treatment

Immediate operation If knife or foreign body still in the wound do not remove it until the field is exposed and suture can be carried out (page 494)

## PERIPHERAL CIRCULATORY FAILURE
### (Shock 'Collapse)

### Causes

1 Diminished blood volume with a subsequent decrease in the cardiac output following
  Hemorrhage
  Trauma with leakage of fluid into tissues of the injured area thus decreasing venous return
  Diarrhea
  Vomiting
2 Increase in the vascular bed (?) resulting from the action of toxins (vasodilators) in pneumonia typhoid etc
3 Neurogenic This type has more acute onset and is usually less serious It is caused by decrease in the vascular tone and reflex cardiac inhibition (vagus)

### Symptoms

1 Patient apathetic
2 Pallor (blood lost or pooled in other areas)
3 Eyeballs sunken features pinched (fluid loss)
4 Temperature subnormal The extremities are cold
5 Low blood pressure (80/60)
6 Pulse is weak and rapid
7 Heart sounds are weak
8 Respirations are slow

### Treatment

**Depends on factors operative in each case**

1 Keep the patient warm The head should be low
2 Give enough morphine to prevent restlessness and relieve pain
3 Transfusion using hypertonic or colloidal solutions Blood is ideal As a substitute use two to four per cent saline or five to ten per cent glucose Acacia has been recommended
4 Drugs of vasoconstrictor group useful in neurogenic shock
   a Epinephrine hydrochloride One cc of 1 1000 solution (subcutaneously)
   b Ephedrine hydrochloride can be given by mouth in doses of 0 048 Gm (3/4 grain)
   c Pituitrin (surgical) one cc (subcutaneously)
   d Caffein with sodium benzoate (U S P XI) given subcutaneously in 8 grain doses is occasionally of value

**Note** Digitalis is contraindicated and should never be used since the rapid heart rate is compensatory owing to the diminution of the blood volume

# 19

# PHYSIOTHERAPY IN THE TREATMENT OF HEART DISEASE

It is part of the cure to wish to be cured —SENECA, *Hippolytus* 1, 249

The use of physiotherapy in the treatment of cardiac patients is not a new concept In the days of Hippocrates physicians were skilled in these measures, and the location of the temples showed their regard for the healing powers of Nature The ancient Celts and Romans recognized the virtues of the mineral springs and advocated hydrotherapy Many Roman coins and other objects which support this belief have been found in the springs at Bad Nauheim Spas were fashionable in Smollett's England and the sharp sighted Matthew Bramble in Humphrey Clinker gives a vivid summary of his opinion of the resorts of his time, All these places have their vogue and then fashion changes" This remark is equally true at the present time although the Matthew Brambles of our day do not suffer from rheumatism and do not travel in coaches Coronary disease has become a more common affliction in an age when the transport plane replaces the coach

The physiotherapeutic measures that have been recommended in the treatment of cardiovascular disease are many and varied, but are either little known or infrequently made use of by the general practitioner Smollett's remark I have neither time nor opportunity to confirm by experiments the particular notions I entertain concerning the efficacy of these waters— [347] can be applied equally well to his professional brethren almost 200 years later This is unfortunate since the chronic sufferer from heart disease often believes his problems are being inadequately managed when something active is not being done for him and many types of cardiac patients turn to the cults where activities of all forms thermal electric and manual are much in evidence The patient consequently passes out of the hands of the physician who possesses a knowledge of the etiology functional capacity and treatment of heart disease to be taken care of by the member of a cult who usually does not know the indications or contraindications for the various methods he employs

The limited training in physiotherapy that the modern physician receives while in medical school probably accounts for the lack of facilities in most hospitals and communities throughout the country The average physician does not know when and to what extent he should take advantage of physiotherapeutic methods in the treatment of chronic disease However, even if he is alert in this respect, there are today few places in

our country where he can send patients with small incomes The resorts and the elaborate establishments are still luxuries that most patients can not afford It remains for the local or state authorities to provide suitable sanatoria where patients of limited means can receive such treatment Perhaps then physicians interested in this work may have time to confirm by experiments prevailing opinions on this form of therapy

## SPA TREATMENT

The spa treatment provides physiotherapy with diet and rest and this accounts largely for its success It is this combination of the spa and its mineral springs with their endless supply of naturally charged water that attracts the patient for it is doubtful if physiotherapy in itself has much appeal to the average patient Consequently we should consider at the start the location organization and equipment of the spa This knowledge of the modern health resort should be familiar to every physician who allows his cardiac patients to undertake the journey seeking the cure for the best results are obtained when the indications and contraindications for this form of therapy are kept in mind

There is a time during the course of treatment of every cardiac patient who is either convalescing from an attack of congestive failure or approaching the end of a rest period following a coronary occlusion, when surroundings become irksome routine medicines sicken and dissatisfaction with life the family and local doctor becomes quite evident Every physician recognizes this phenomenon His patient is not ready to resume his customary duties but is able to increase his activities and venture beyond the confines of the bedroom for the first time The very chronicity of the cardiac ailment and the chorus of don ts from family and doctor discourage the patient and make him yearn for a change At this point in the management if finances permit properly supervised spa treatment may prove very helpful

The spa is the Shangri La of the modern business man He enters upon a new regime of treatment with enthusiasm and life takes on a rosy glow He is removed at once from daily contact with motor noises exhaust gases traffic snarls jangling telephones city apartments radio loud speakers night clubs and well wishing friends and he enters upon a period of training at the spa where something is being done for him every minute of the day Attendants hop bands play and it is small wonder that his lagging spirits rise The initial circulatory stimulant is purely psychic but none the less beneficial The patient meets other sufferers from similar ailments and in their company he willingly follows the plan of treatment devoted to cardiac preservation In this cheerful setting where nothing is lacking to provide comfort and happiness he is taught to live within the limits of his reserve If the spa cannot cure, it can educate and IT IS JUST AS IMPORTANT TO TRAIN THE CARDIAC PATIENT IN HABITS OF LIVING AS IT IS TO TRAIN THE DIABETIC IN HABITS OF EATING

**The success of physiotherapy at the spa** depends a great deal on the proper selection of cases At the beginning of the present century Mackenzie in plain language pointed to the folly of physicians who allowed cardiac cripples totally unfit for travel to undertake long journeys to the spa for the cure He coined the term 'Nauheim wrecks for patients who returned much the worse for the experience The physiotherapeutic measures used at the spa are most beneficial in ambulatory cases from Therapeutic Classes A B and C In other words, patients with evidence of congestive cardiac failure severe cardiac asthma angina on slight exertion recent or large occlusions advanced syphilitic heart disease subacute bacterial endocarditis and active rheumatic carditis do better at home under the care of the family physician Patients who have mild angina obesity hypertension and effort syndrome as well as sufferers from peripheral vascular disorders particularly of the spastic type the coronary occlusion convalescents and patients with inactive rheumatic disease and diminished cardiac reserve may derive some benefit from a course of Spa *treatment*

From the description so far it is evident that much of the cure depends on the environment and the attendants and may be classed as psychic Consequently in the following attempt to sum up the benefits of the different forms of therapy we must admit that there are many advantages that cannot be measured by the yardstick of science

## HYDROTHERAPY

Hydrotherapy (Balneotherapy) is usually administered as a carbon-dioxide bath This form of therapy in cardiovascular disease was originated at Bad Nauheim in Germany and such favorable results have been reported that other centers have been established where naturally carbonated waters are available in large quantities Well known spas are located at Bath and Harrowgate in England Royat and Vichy in France Bad Nauheim in Germany and Saratoga Springs Bedford Springs, Hot Springs White Sulphur Springs and Palm Springs in America

**The carbon dioxide bath** is prepared from mineral waters which emanate from the earth When this water reaches the surface great quantities of carbon dioxide escape but the bath retains a high percentage of the gas in solution a fact that can readily be demonstrated by observing the large amount of carbon-dioxide bubbles that cling to the body surface of the patient immersed in the bath (Fig 160) The amount of carbon dioxide in the water as it comes from the earth averages 0 7 to 1 4 Gm per liter although it is higher at some spas Different springs at the same spa sometimes vary in their carbon-dioxide content The water from these springs is always alkaline in reaction

The temperature of the water as it issues from the earth should not be too high in order that enough carbon dioxide may be retained in solution The ideal temperature has been found to be between 97° and 99° Fahren

heat However many resorts such as Royat have springs of varying temperature permitting a wide choice in prescribing a natural bath to suit the needs of the individual patient

The effect of hydrotherapy on the cardiac patient has recently been studied by a number of observers[39-41] The mechanical effect of the bath itself the temperature at which it is given and the chemical effects of the carbon dioxide on the bodily processes locally and after absorption, are the main facts to be considered in an evaluation of this form of therapy

It is a common observation that the skin of the patient in the carbon dioxide bath becomes hyperemic up to the level of immersion This is at-



FIG 160 Bubbles of natural carbonic acid gas blanket the whole body in the baths (Courtesy Dr Walter S McClellan Saratoga Springs New York)

tributed by some to the direct vasodilating action of the carbon dioxide on the skin vessels Consequently during the first few minutes in the bath the patient experiences a pleasing sensation of warmth even if the temperature of the water is below 99° F and an immediate effect is reflected in the blood pressure A gradual drop in the systolic as well as the diastolic levels occurs the peak is reached in about ten minutes In some patients a 30 to 40 mm decrease in the systolic blood pressure is not unusual If the patient stays in the bath the pressure will be observed to rise again and will reach its initial level in about ten minutes It is claimed that as the number of baths increases there is a tendency for the pressure to remain at lower levels in certain groups of patients and this is accompanied by an increase in the oscillometric index However we must always be careful in interpreting results in terms of the blood pressure reading that is so easily influenced by a variety of psychic factors The blood pressure would naturally tend to decrease in some cases in the atmosphere of the spa amid the pleasant surroundings and in the absence of disturbing contacts

During immersion in the bath the pulse rate drops and according to

some observers an actual diminution in cardiac size may follow the treatment

The pressure of the water of the bath may likewise aid in venous emptying Intra abdominal pressure may be increased by the weight of the water and this may augment the volume of venous blood returned to the right side of the heart Kroetz and Wachter have shown that in the baths at Nauheim there is an increase in the minute volume output of the heart The increase is greater in the carbon-dioxide bath than in the plain or fresh water baths It is also claimed that the respiratory center is stimulated directly by the increased amount of carbon dioxide in the arterial blood during the carbon-dioxide bath This may be caused by the increased amount of the gas absorbed from the skin and eliminated through the lungs or carbon dioxide may be inhaled from the environment In any event the amount of carbon dioxide eliminated by the lungs during and following the bath increases [91] Moreover the oxygen consumption appears to be only slightly greater certainly not to the extent that would indicate that the carbon dioxide comes from the oxidative changes of metabolism

In spite of these studies the exact mechanism of the action of the carbon dioxide remains unsettled The observers who support the theory of local action claim that the whole effect of the bath results from the stimulation of the cutaneous nerve endings Marked vasodilatation occurs the reflexe cause slower heart action The repetition of the baths accounts for the good effect in certain spastic arterial diseases of the extremities The proponents of the absorption theory claim that the gas enters the body in solution and acts on the organism as a whole increasing respiration by action on the respiratory center in the medulla The excess carbon dioxide in the mineral water causes peripheral dilatation and this again allows more carbon dioxide to pass through the skin Some observers carry the theory a step further They claim that the carbon dioxide acts as a sedative for vagosympathetic excitability and that it stimulates nutrition by improving the basal metabolism and helping in the excretion of toxic products In their opinion this explains the beneficial effect in hypertension A diuretic action has been said to be another good effect brought about by the carbon dioxide bath through *its increase in the basal metabolic rate*

The improvement in the morale may explain the good results that follow spa treatment in many of the coronary cases particularly in the patients who have mild degrees of angina Many angina cases especially if hypertension is present are apt to show periods of temporary improvement under any new forms of therapy A study of the electrocardiogram before and after the usual regime of spa treatment in 107 cases is offered as evidence of improvement by Comstock and his associates [70] However it must be realized that in no condition is the electrocardiogram subject to so much variation as in coronary disease particularly in patients recovering from minor occlusions

Considering the various effects of the carbon-dioxide baths that I have outlined, both real and theoretical the first indication for this type of treat

ment seems to be certain forms of hypertension where the secondary changes have not been too widespread or rapid but where the symptoms have been persistent and annoying Whether or not we believe the claims made for carbon dioxide baths any therapy that proves useful in calming the dangerous crises encountered in the hypertensive group and allows periods of relief to punctuate the course of a disease acknowledged to be chronic should be used even though we cannot analyze every phase of its action I do not believe that the effect of the carbon-dioxide baths is at all lasting in the majority of cases of established essential hypertension but I do believe that their use combined with other measures at the spa is far better treatment for this group of patients than any other system of therapy in the absence of symptoms of cardiac failure

While it is unfortunate that the result of the carbon-dioxide baths is regarded in terms of so many millimeters of fall in the blood pressure by both patient and physician on the other hand observations of the blood pressure are a great advantage in selecting cases The patients who show a moderate drop in blood pressure accompanied by a feeling of well being after the first carbon dioxide bath are usually the ones who will show the most benefit from a series of these treatments If the blood pressure seeks its former level when the patient returns home however he should not be discouraged for this does not indicate that the stay at the spa has been in vain If the onward march to ultimate cardiac failure has been at all delayed by the rest program that accompanies the treatment much can be said in favor of the spa Likewise if the cardiac patient returns from the spa with his mode of living adjusted to a more moderate level he should not worry about his blood pressure readings He should realize that many of his annoying disturbances such as vertigo headaches and flushes have been at least temporarily benefited by the treatment

The spa regime should prove of great value to the obese hypertensive who has the ruddy glow of health since it combines dietary regulation with hydrotherapy and the proper prescription of exercise Careful attention to every detail often makes it possible for the spa physician to uncover and treat successfully some secondary condition this in the long run reacts favorably on the hypertension The thin hypertensive patient who is subject to vascular spasms obtains relief because of quiet environment and the dilating effect of the carbon dioxide baths Much benefit likewise results in all groups of patients when they learn that they can live without having blood pressure readings taken several times a day

I heartily disagree with the claim that reduction of the blood pressure in the presence of aortic insufficiency is a benefit to be derived from the CO cure This form of hypertension is a compensatory mechanism and in itself is certainly no indication for therapy Likewise the hypertension associated with an advanced renal lesion and early renal decompensation is not the type that is amenable to vigorous spa treatment When carbon dioxide baths are said to have ameliorated the renal lesion eliminating the albuminuria and decreasing the blood pressure level and nitrogen re

tention I always wonder what the patient s course would have been with out the special therapy, since renal lesions many times display quiescent periods

The sedative action of the carbon dioxide baths may be counted on to help patients with overactive sympathetic nervous systems particularly those who show little if any basic cardiac damage and who are constantly plagued by frequent premature beats The same may be said of patients suffering from frequent paroxysms of tachycardia where the usual drug therapy is either poorly tolerated or ineffective in preventing recurrence of the seizures Patients with the tachycardia of mild hyperthyroidism may take the cure if financially able Surgery however offers the poorer patient a much quicker return to health Physiotherapy is contra indicated in advanced hyperthyroid states with cardiac signs and symptoms However during convalescence following surgical treatment physiotherapy may be useful

To give the reader an idea of the number temperature and types of baths administered the following case histories from a recent article by McClellan [44]* showing the course of hydrotherapeutic treatment advised in each instance at Saratoga Springs are included

## ILLUSTRATIVE CASES

**Case 106** H M male age 75 was referred by his physician to Saratoga on April 9 1935 Some dyspnea on mod rate exert on and claud cation in the lower extr mit es on walking short distances were present for some years

PHYSICAL EXAMINATION The patient was above the average height thin and distinctly nervous It was d fficult for him to sit still during the examination There was arcus senilis and slight pulsation of the larger ves els of the neck His heart was slightly enlarged to percussion and the puls rate was 95 There were numerous premature beats occurring at irregular intervals The blood pressure was 152 systolic and 90 diastolic The radial arteries were palpable and not easily compressed The lungs were clear Abdom nal examination was essentially negative In the extremities the reflexes w re present and weak pulsations could be detected in both dorsalis pedis arteries There was slight pitting edema about the ankles

*CLINICAL DIAGNOSIS* A *Etiologic Arteriosclerosis* B Anatomic Card ac enlargement C Physiologic Premature ventricular contractions D Functional Clas ification Class 3

COURSE OF TREATMENT The following table presents in detail the temperature duration and position of the patient in the bath as well as a record of his puls before during and after each bath

| | Bath Prescription | | | Pulse | | |
|---|---|---|---|---|---|---|
| Date | Temperature F° | Duration Minutes | Full ¾ or ½ Bath | Before | During | After |
| April | | | | | | |
| 9 | 95 | 8 | 3 | 90 | 88 | 85 |
| 10 | 95 | 9 | | 85 | 84 | 83 |
| 12 | 95 | 10 | | 85 | 87 | 80 |
| 13 | 94 | 10 | | 84 | 92 | 80 |
| 15 | 94 | 10 | | 84 | 91 | 78 |
| 16 | 94 | 10 | | 85 | 80 | 78 |
| 17 | 94 | 10 | | 83 | 79 | 76 |

* Reprinted by permission of the author and the J B Lippincott Co

| | Bath Prescription | | | Pulse | | |
|---|---|---|---|---|---|---|
| Date | Temperature F° | Duration Minutes | Full 3/4 or 1/2 Bath | Before | During | After |
| April | | | | | | |
| 22 | 94 | 10 | Full | 73 | 78 | 78 |
| 23 | 94 | 10 | | 77 | 77 | 78 |
| 24 | 94 | 11 | | 78 | 76 | 77 |
| 26 | 94 | 12 | | 77 | 76 | 77 |
| 27 | 94 | 12 | | 78 | 77 | 76 |
| 29 | 94 | 12 | | 78 | 76 | 75 |
| 30 | 94 | 12 | | 79 | 75 | 74 |
| May | | | | | | |
| 2 | 94 | 12 | | 78 | 74 | 78 |
| 3 | 93 | 12 | | 78 | 75 | 77 |
| 4 | 93 | 12 | | 8 | 74 | 76 |
| 6 | 93 | 12 | | 77 | 75 | 74 |
| 7 | 93 | 12 | | 76 | 74 | 75 |
| 9 | 93 | 12 | | 75 | 74 | |
| 10 | 93 | 12 | | 74 | 73 | 76 |

Progress Notes April sixteenth The patient while still nervous has less edema about the ankles He states that pain in the legs is less severe The heart rate is 85 and frequent premature beats are still present

April 23 Symptomatic improvement is evident and the patient states that he can walk with less dyspnea and pain than at the beginning of his treatment

May Th re is still very slight edema at the ankles The pulse rate is 78 with the irregularity persistent

May 10 Pulse rate is 4 He states that he feels distinctly better There is no edema of the ankle although the cardiac irregularity still persists He is able to walk approximately one mile without the production of pain in the lower extremities

May 16 A letter from his physician written after his return states that both objectively and subjectively this patient is much improved and he is advised to repeat the course of treatment at the end of six months

Discussion This patient's history is typical of the gradual progress of a widespread arteriosclerosis The pain in the lower extremities was apparently an expression of diminished blood supply caused by the general arterial disease We do not expect that a patient of this type will show any decrease in the amount of arterial change or that the progress of the condition will be arrested The treatment however is of value in improving the circulation by making full use of the blood channels which are still available This type of patient does well on courses of treatment once or twice a year in order that the improvement noted above may be maintained The additional rest periods procured by visits to the spa likewise contribute in no small measure to the symptomatic relief obtained

**Case 107** R G male 46 years referred to Saratoga on July 2? 1935 by his physician who wrote that the man had been able to carry on his work in his usual manner until the past April when he had experienced a sudden sharp pain in the precordial region which had been sufficient to require bed rest The first attack of pain occurred in April 1935 and had been followed by a second attack on May 9 1935 The clinical examination and electrocardiographic tracings supported the diagnosis of coronary occlusion The physician stated that since the attack the patient had been at home and unable to perform any work although he had been up and about the house for the past six weeks and able to walk one or two blocks

The examination at Saratoga showed a well developed and well nourished middle

aged man Heart rate 78 regular both sounds distinct and no murmurs Blood pressure 132/80

CLINICAL DIAGNOSIS A Etiological Arteriosclerosis B Anatomical Coronary sclerosis and old occlusions C Physiological Normal sinus rhythm D Functional Classification Class 3

TREATMENT The patient took the course of carbon dioxide mineral water baths shown in the following table

PROGRESS NOTES It will be noted that after the first bath there was an elevation in the rate of the pulse which may be accounted for by the nervous reaction following a new type of treatment

| DATE | BATH PRESCRIPTION TEMPERATURE F° | DURATION MINUTES | FULL ¾ OR ½ BATH | REST | PULSE BEFORE | AFTER |
|---|---|---|---|---|---|---|
| July | | | | | | |
| 22 | 93 | 8 | ¾ | 1 hr | 72 | 86 |
| 24 | 93 | 9 | | | 66 | 64 |
| 27 | 93 | 10 | | | 72 | 60 |
| 29 | 93 | 10 | | | 0 | 63 |
| 31 | 92 | 10 | | | 80 | 63 |
| August | | | | | | |
| 1 | 92 | 10 | Full | | 72 | 63 |
| 3 | 92 | 11 | | | 72 | 60 |
| 5 | 91 | 11 | | | 72 | 66 |
| 7 | 91 | 11 | | | 72 | 62 |
| 8 | 91 | 12 | | | 72 | 60 |
| 10 | 91 | 12 | | | 80 | 72 |
| 12 | 91 | 11 | | | 72 | 72 |
| 14 | 90 | 11 | | | 72 | 64 |
| 15 | 90 | 12 | | | 72 | 72 |
| 17 | 90 | 12 | | | 72 | 72 |

August 1 Patient states that he is able to walk five blocks without the production of any pain at the present time He is anxious to take up his work and it is difficult for him to follow a course of complete rest at home

August 17 The patient feels that he has distinctly gained in strength and is now doing light work around his home such as caring for the lawn and some light carpenter work His pulse rate is steady at 7 good quality and his blood pressure is normal

DISCUSSION This patient undoubtedly had a good result following the period of spa treatment He is a typical example of the problem presented by the active business man convalescing from a coronary occlusion who experiences anginal attacks when first allowed to be out of bed The period of training the patient procured at the spa including the $CO_2$ baths was far more effectual in bringing about his return to work than drug therapy

There are additional features offered by the spa treatment that in my opinion far outweigh the value of the waters In this instance the spa was useful when placed between hospital and home At the spa this patient acquired the proper philosophy toward restricted living which an immediate return to the home and business would not have permitted He was given a good chance at the spa to become acquainted with his myocardial reserve and he did this with better grace when placed in the company of those similarly incapacitated The baths were prescribed but the routine rest periods that followed the treatments gave them their greatest value

Not the least of the benefits that some patients have shown following their visits to European Spas has been derived from the voyage over and back two enforced periods of physical and mental rest

## MASSAGE

Massage is another valuable measure that is commonly overlooked in the management of the cardiac patient The layman has always been awake to the value of this form of physiotherapy but as the physicians grew in scientific knowledge massage was unfortunately entrusted to others Continued neglect and lack of proper supervision gave opportunity for the rise of the various cults These less informed enthusiasts have gained in confidence and boldness until they claim to be able to cure almost any disease by massage

Massage has been shown to produce immediately dilatation of the capillaries the duration of which depends on the amount of pressure used [8] This effect can be observed by the direct inspection of the capillary bed with the miscroscope In addition the red blood-cell count is increased by forcing inactive cells into the circulation from resting areas Light massage can be useful even in cases of mild cardiac failure since it aids the return of venous blood and in long periods of inactivity makes up for the lack of the force supplied by contraction of the leg muscles Massage properly carried out therefore is an aid in preserving some degree of muscular tone and vigor

Massage is particularly valuable if given following the application of heat This principle is used in the Vichy douche where the entire skin area is sprayed by showers of different temperatures and then massaged After all these treatments whether local or general the cardiac patient should rest since the procedure is usually followed by a sense of fatigue In severe congestive failure with a high degree of venous engorgement massage is contraindicated It is likewise good policy to defer its use in the presence of fever acute endocarditis or acute myocarditis

## HELIOTHERAPY

Many of the spas have special rooms fitted with equipment for combining the effect of ultraviolet light with other forms of therapy In America the quartz lamp has invaded the hospital the athletic club the sanatorium the physician s office and lately the private home Much benefit is claimed from its use in various forms of heart disease including angina At present the subject is in a very unsettled state and further investigations are needed before definite conclusions can be made It may be a great aid in the treatment of children with rheumatic carditis (Fig 161) as we have long known that this disease flourishes in the crowded slum areas of the large cities where there is lack of sunlight and shows

decreasing incidence in the warmer climates of the South where the amount of sunlight is greater

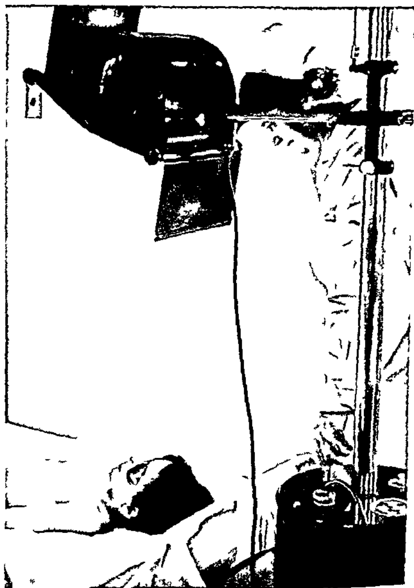


FIG 161 Heliotherapy (Courtesy Children's Heart Hospital Philadelphia)

## ELECTROTHERAPY

The value of the various electrotherapeutic procedures in the treatment of cardiovascular disease is still unsettled Further studies are essential to

support the claims that have been made for diathermy high frequency static sinusoidal galvanic and faradic currents Autocondensation and diathermy are most popular in the treatment of hypertension Diathermy undoubtedly has some place in the treatment of cardiovascular conditions where heat relieves the symptoms The greatest benefit follows its use in arterial diseases of the spastic type and intermittent claudication The treatments likewise have some temporary effect in relieving symptoms of other conditions In coronary disease accompanied by angina if the element of spasm predominates diathermy may be helpful by producing vasodilation and increasing coronary flow Some cases are reported where more lasting benefits have resulted than followed the use of the nitrite drugs In these instances the short wave diathermy has been thought to produce its good effect by a temporary paralysis of the sympathetic nerve pathways However in all these cases the psychic element is difficult to rule out

**Hyperpyrexia** The production of hyperthermia or hyperpyrexia by the use of the Kettering or similar apparatus has been followed by encouraging results in some cases of infection of the heart Cures have been reported occasionally in acute gonococcal endocarditis (page 189) and here the use of the method seems to be a rational one since it is quite possible to produce temperatures in the human body that the gonococcus cannot withstand

In chorea the use of the Kettering apparatus has many advantages Although great care should always be taken in the presence of an active carditis in recommending this form of therapy for children recent successes that have been reported are most encouraging It must be remembered that artificial fever is different from fever of toxic origin it is produced by electromagnetic induction and is not a fever in the ordinary sense but a heating of the body a hyperpyrexia Neymann [3] reports excellent results in a series of 25 cases of Sydenham s chorea In all cases the choreiform movements stopped following the treatments Similar results have since been reported by other investigators Neymann recommends that the treatments be given bi weekly, and that the temperature at each treatment should be maintained between 39 7° C (103 5° F) and 40 6° C (105 F) for eight hours After treatment the cardiac rate is increased and the T waves may be flattened but these changes disappear in 24 hours This form of therapy is not beneficial in all forms of chronic carditis and is not to be recommended for general use

## OCCUPATIONAL THERAPY

The proper form of occupational therapy can only be prescribed when a complete diagnosis of the patient is at hand and the functional classification determined consequently it is one of the final considerations in treatment Patients placed in Functional Group 4 can handle only the types of occupational therapy that can be carried out at bed rest Pa

tients convalescing from repeated attacks of congestive failure and the rheumatic groups with cardiac infection make up this category Although these cases are allowed very little exertion the mental rest that follows interest in some task that can be carried out for a few hours each day is a considerable aid to children and adults alike If improvement permits advancement to Group 3, and finally to Groups 2 and 1 the value of occupational therapy as a future means of earning a livelihood becomes important Some form of physiotherapy that is begun to interest the patient for a few hours a day may ultimately prove to be

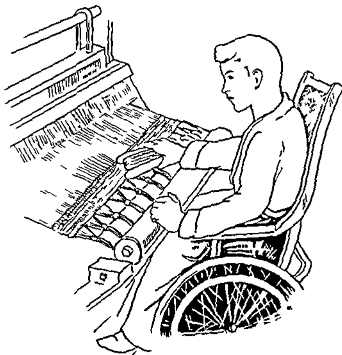


Fig 16 Occupational therapy

an excellent source of income Less financial worry often results and this is reflected in the health of the patient I recall an insurance salesman of 52 who came into the hospital on two occasions following attacks of coronary occlusion He showed such limitation of cardiac reserve after the second attack that he was forced to give up his work He became much interested in the manufacture of sets of picture puzzles then at the height of their popularity When he left the hospital after two months he was improved but was still in Class 3 However he already had ideas for enlarging the business and with the help of his wife and daughter, who made up his sales force he was soon able almost to equal his former income This patient had an excellent outlook did not talk about his condition any more than it was necessary at the time of the visits of his

physician and never had time to realize that he was a chronic invalid He learned how to use his hands in a new occupation that allowed him to spare his heart Many other cases where the morale of the patient has been improved through the use of occupational therapy could be cited (chapter 23)

For Class 3 and 4 patients confined to bed various ingenious devices have been arranged to permit tasks requiring the use of the fingers only Light bed boards may be used or in some cases where weaving is carried out the apparatus can be suspended over the bed (Fig 162) Prolonged hospitalization permits training for occupations which require extensive apparatus while patients who come in for shorter periods may be assigned to simpler tasks like basketry cord and leather work Women patients may in addition to any of these types of occupational therapy renew their interest in different forms of needlework

Even in shorter illnesses the psychotherapeutic value of some slight task assigned cannot be overlooked The patient may be inclined to worry about his condition the new surroundings may delay his convalescence hence attempts should be made to interest him in some occupation as soon as his physical state permits In this way prolonged hospitalization may be prevented needed articles may be produced for the supply room (folded surgical dressings sponges etc ) and the patient s morale may be kept at a high level

# 20

## THE PRESCRIPTION OF EXERCISE

The wise for cure on exercise depend'

—DRYDEN, *Epistle to John Driden* 1 94

Exercise plays an important role in the management of the cardiac patient Unfortunately rest has been emphasized so much that patients are instinctively afraid of exercise, and for this reason physicians are apt to prescribe exercise cautiously and vaguely, if at all The prescription of exercise should present little difficulty if the physical examination has been made with care and the functional capacity determined The advice given to cardiac patients regarding their activities is based on this data as well as on the principles underlying the physiology of exercise

Energy for exercise is furnished by ingested food From a resting level when the requirements may be but one calorie per minute to the exercise level when the requirement may be ten times as great it can be seen that the resources of the body must be drawn upon to supply the energy as it is needed Analysis of the urine after the most severe exercise shows such a small amount of extra nitrogen that we know that the protein stores have been but little depleted Intelligently arranged training tables for athletes, therefore are not any longer overburdened with meat and other nitrogen containing foods, since the energy for muscular contraction is more quickly derived from the breakdown of substances that are non nitrogenous This is shown by a study of the oxygen absorbed and the carbon dioxide eliminated (respiratory quotient) The carbohydrates of the food furnish most of the motive power for muscular exertion Consequently athletes are now encouraged to eat candy before engaging in severe muscular activity

As a result of muscular contraction there is an accumulation of lactic acid which disappears rapidly in the presence of oxygen The chemical changes in the blood accompanying exercise are responsible for stimulation of the respiratory center There is likewise increase in the pulmonary blood flow proportional to the increase in ventilation, a rise in the output of the heart per minute and an accelerated heart rate The size of the heart of the normal athlete who is well trained is unchanged after severe exertion but the blood pressure shows a temporary increase This is more pronounced in the athlete because of the psychic factors of competition and is brought about by the constriction of the splanchnic area It permits widening of the capillary beds in the heart and skeletal muscles and aids in the quick diffusion of oxygen to essential areas

Although the presence of oxygen is not necessary for contraction of the

muscles it is most essential for their recovery A person can perform a given task without the necessary amount of oxygen being present for its full accomplishment For example he can hold his breath and climb a steep grade or lift a heavy weight but to do this the tissues go in debt for oxygen Our ability to proceed with the tasks requiring the sudden expenditure of energy without the appearance of symptoms depends upon this physiologic fact The debt of oxygen is paid back by a continued elevation of the respiratory rate following exertion for a longer or shorter recovery period until the balance is restored

The training of an athlete to accomplish a task with increasing efficiency and skill depends on daily practice This eliminates awkwardness and unnecessary movements and lessens the amount of oxygen needed each time The size of the skeletal muscles precipitating in the particular task is increased and the bodily processes of taking in the oxygen (respiration) and distribution of the oxygen (circulation) become better regulated and adapted The vital capacity increases as training progresses Consequently graded exercise may build up a reserve that enables the ultimate performance of a task to be accomplished with few if any distressing symptoms In a similar manner the cardiac patient can train his depleted reserve to perform more efficiently within narrower limits Therefore in neglecting to furnish the proper prescription of exercise the physician is not making use of all possible aids to recovery

Restriction of activity on inadequate grounds will consequently do more harm than overlooking a slight organic lesion and permitting full activity The mental effect of telling the patient that he has a heart murmur is tremendous particularly if it is followed by vague advice to take things easy for a while (chapter 13) A fear may develop toward all forms of activity and the subsequent lack of exercise soon reflects itself in a poor physical state Therefore it is most essential that the physician be fully acquainted with the physical condition of the patient before prescribing or prohibiting exercise

Let us consider first the types of heart disease where bed rest for prolonged periods is required In this group we include patients with congestive failure recent coronary occlusions and active cardiac infections Even in these instances active exercise in all forms is not contraindicated beyond a few weeks In exceptional cases even before this period has expired some forms of exercise may be definitely indicated to encourage the patient and keep up his morale Nurses trained in the care of cardiac patients are valuable at this time They help to take the patient s mind away from his condition and interest him in some form of exercise even if it consists only in moving the fingers in arranging cards or in making small articles Gentle massage may be started almost at once in many patients and this may be followed by passive movements If no ill effects are noted greater freedom may be allowed The Schott resistance exercises if properly carried out are often valuable in beginning the rehabilitation

When to allow the patient out of bed is a decision that rests on the facts of each case Experimental evidence demands that the rest period for the patient following an acute coronary occlusion should be not less than six weeks However, patients who show good recovery from congestive failure may be permitted to be out of bed much sooner When edema disappears and the appetite improves it is often good policy to allow a little freedom as early as the end of the first week It has been demonstrated many times that nothing is to be gained by unduly prolonged bed rest, particularly in cardiac patients at or beyond middle life Furthermore many complications are encouraged by an extended period of inactivity in elderly individuals

Walking is the next exercise that may be prescribed for this group the amount depending on the effect of the initial allowance on edema chest pain pulse, and respiration In the patient who has angina the increase in the exercise allowance should be gradual If chest pain is absent the prescription may be slowly and cautiously increased for it is likely that no harm is being done

The same rule governs the first time the convalescing cardiac patient goes down stairs or out-of-doors It is not harmful to descend a stairway for this in itself is a form of self administered resistance exercise and in addition may help to restore the sense of balance after a period of bed rest The return trip however should be taken slowly 'One step at a time is trite advice but it still holds

In prescribing the first walks outdoors tell the patient in definite terms how far he is to go The importance of graded walks at this stage of convalescence has long been recognized and is part of any well planned spa regime The most elaborate of the systems is the one proposed by Stokes and Oertel consisting of a series of carefully laid out walks the first covering a distance of 1/12 of a mile While walking this distance the patient lifts his weight up a 3 per cent grade If the response is good the grade is gradually increased and over the course of some weeks the patient will be able to cover a distance of two miles comfortably with the grade advanced to 15 per cent An elaborate course for this system of exercise was laid out some years ago at Hot Springs Arkansas Foot paths appropriately labelled by stone markers of varied hue lead the convalescent through scenes of natural beauty They are graded from almost level to very steep A truly American adaptation of the Oertel system may be found at Saratoga Springs where the patient takes his daily amount of prescribed walking through a carefully planned golf course

In obese patients this system of exercise is very valuable when combined with the proper dietary regime Over the course of eight weeks' treatment by diet and walks considerable improvement in exercise tolerance can be noted The more drastic forms of Swedish exercise carried out with the aid of gymnasium equipment, are less popular today but they are still useful when carefully prescribed The stationary bicycle and the rowing machine in the city gymnasiums and clubs furnish satisfactory forms

of indoor exercise for the winter months Intelligent supervision is much more important however than the type of exercise The response of the patient to each added burden is the essential factor for this suggests the amount to be added or subtracted from the next day s exercise pre scription

Less fortunate patients who cannot take spa cures' may nevertheless derive benefits from walks if the physician will take the time to plan them Familiarity with the neighborhood of the patient s home or hospital will make this exercise prescription possible In addition to keeping alive the patient s interest in his treatment and progress the physician s notations concerning these exercise prescriptions show the functional capacity and when considered as a whole they are invaluable in estimating the effect of therapy as well as the ultimate prognosis in each case

Before planning suitable exercise for an ambulatory cardiac patient a knowledge of the occupation and temperament is essential I have never advised the so-called setting up exercises for they are tiresome in most instances and after the first few attempts are usually abandoned The ideal forms of exercise for cardiac patients are the intermittent and non competitive types Golf for example is excellent if the patient s tastes run in this direction and a prescription of nine holes will be easy to accept This takes the patient outdoors away from business and adds the effect of heliotherapy for this reason it is far better than a gymnasium Further more if the patient is interested the golf is more apt to be continued Golf should not be attempted by those who are not interested in it or where competition cannot be avoided or if emotional strain is increased

The physician of today should have a fair knowledge of the different types of out of door activities if he is to prescribe these forms of exercise successfully Tennis and similar competitive games should not be allowed Croquet and other lawn games may be suitable as far as exercise goes but may be unfit as far as the personality of the patient is concerned Swimming if properly supervised is suitable in a number of cases The convalescing cardiac patient will usually prefer to take his allowance of exercise in the morning hours for then it does not interfere with sleep Wakeful nights are more apt to follow exercise periods taken at the end of the day

The ambulatory patient seen at the dispensary will have neither time nor money to follow any special program for exercise The question of exercise in these instances may be considered in relation to the usual type of work Cardiac patients are happier when permitted to return to work as soon as possible Exercise should be planned with this in view but before the schedule is made out the physician must have an under standing of the type and amount of exertion which the patient s daily work entails While lighter factory work may be permitted heavy laboring jobs are out of the question and here the great problem arises In days before the depression when opportunities for new positions were more plentiful the problem was usually settled satisfactorily by the Social

Service department (chapter 23) Today it is more difficult for the cardiac cripple to become self supporting but I have seen many patients who have demonstrated that it is not impossible Occupational therapy begun as a mild form of exercise and mental diversion may jump to the fore as a means of livelihood, since useful articles manufactured in convalescence may find a market later

As a general rule the cardiac patient with few exceptions should be encouraged to engage in some form of activity The complete retirement made possible by an insurance policy is not always a blessing and does not necessarily carry with it a guarantee of a longer life The attitude toward life that these patients sometimes adopt is by no means the best The lack of exercise is soon evident and if they are compared with the compensated cardiac patient of the same age with the same lesion who is physically fit, the difference is striking

In cases where the patient holds a more responsible position in the business world adjustment is much easier An earlier return to work may be permitted if a volunteer can be secured who will drive the patient to and from his office if this is at a distance With the question of transportation settled the hours of work can be adjusted according to the reaction It is better to start with a prescription of two hours and increase the working hours gradually Later with improvement short walks or the way to work may be added A few city squares in the early morning may lack the stimulating surroundings of the spa but after all it is the state of mind of the patient that influences the outcome of the battle This can be moulded and directed in large part by the physician during the early days of the illness

If the patient is of school age there are many questions to settle in regard to the exercise allowance when permission is given to return to classes Careful training of the cardiac child is possible so that the two extremes mentioned by Robinson[319] are avoided The child then neither attempts to cover up the handicap nor uses the handicap to 'cover up' The usual strenuous gymnasium activities should be entirely prohibited, but the milder exercises prescribed for correction of faulty posture may be tried for many times poor posture places an additional burden on the circulation The child should be encouraged to take walks The stairway of the school should not stand as a handicap to the education so essential to these cardiac children The steps can be taken if time enough is allowed The child who has a cardiac lesion should be taught not to run to school at school or after school

Upper respiratory infections are more serious in children because of the likelihood of stirring up a latent rheumatic infection For this reason all activities, including attendance at school, should not be permitted when fever is present It is always the best plan whenever possible to allow the child who is handicapped by a cardiac lesion to attend the special classes that have been organized in some cities for his care Here under the supervision of well trained teachers and in the company of other chil

dren suffering from similar limitations progress may be more satisfactory The children in these special groups or schools are taught to avoid haste in all things and a philosophy of living under a constant restriction of exercise is acquired Even when exercise has to be curtailed for long periods because of the recurrence of rheumatic infection education is still carried on in these special schools In this way the patient is prepared to fill a position in life that will not overtax a decreased cardiac reserve (page 131)

## THE HEART IN ATHLETICS

Nearly a century ago at the beginning of the modern era of collegiate athletics, James Hope in a book entitled Diseases of the Heart and Great Vessels expressed the opinion that boat racing at Oxford and Cambridge and violent gymnastics have caused

> rupture and inflammation of the aortic valves and aorta, issuing in incurable organic disease

He further stated

> I have also known pedestrian tours among the Swiss and Scotch mountains to be followed by hypertrophy and other diseases of the heart It is protracted efforts that are always most pernicious Feats of this kind should always be discouraged [181]

A similar stand was taken by Peacock in 1864 when he claimed that cardiac hypertrophy and subsequent failure among Cornish miners resulted solely from overwork In 1870 Clifford Allbutt claimed that long continued exertion caused right heart failure and dilatation and that sudden strains were productive of wear and tear in the aortic area In 1888 Roy and Adami s experimental work seemed to lend support to these claims and chronic thickening of the cardiac valves due to strain was described at length in their book Adami in 1911 stated that he believed the nodose arteritis seen in elderly subjects was associated with thickening of the valve leaflets both in his opinion resulted from mechanical strain In 1898 and 1909 Allbutt retrenched enough to admit that the influence of the toxic and infective factors was difficult to eliminate in these cases and sounded the first modern note in his conclusion that the importance of muscular effort as a factor in cardiac injury has been much exaggerated

However for years there were no dissenting opinions Reviewing the literature up to 1915 we find the old idea re echoed many times Bardeen[19] in 1915 stated

> All college students taking part in major sports have hypertrophied hearts While in many cases the compensation is good in a large number there is myocardial irritability sometimes accompanied by mitral murmurs which indicate somewhat serious lesions

For the past 25 years, with a few exceptions little attention has been given to the subject in American and English literature An extensive continental literature is available but it contains many conflicting opinions Consequently the subject of the heart and athletics remains rather hazy in the minds of many The prescription of exercise for damaged and undamaged hearts shows a surprising lack of uniformity in the various university centers and the functional systolic murmur continues to consign good athletes to the side lines In these cases even in the absence of hypertrophy or any supporting sign cardiac involvement is suspected, and even the mildest forms of exercise may be prescribed with hesitation and uncertainty

The sustained interest of both sexes in all forms of athletic activity places *the question of the heart in its relation to competitive sport* squarely before the health services of our universities Curiously enough the rising tide of interest in sports and their effect on the heart has been paralleled by revolutionary changes in our concepts of diseases of the circulatory system With Mackenzie and Lewis as leaders a newer cardiology has sprung up in which valve lesion and murmur are now relegated to the background and the heart muscle assumes major importance We have reached the time when the older idea especially the erroneous assumption that the athletic heart is a clinical entity must be discarded and our judgments must rest on the sounder foundations of these newer concepts

To begin with let us consider the normal heart of an athlete at the end of a hard race We look first at the clinical picture We see the runner gasping for breath we note his pale pinched expression and weak rapid pulse He perspires freely from a cold body If the exhaustion is more severe we may see him lying on his side occasionally doubled up complaining of abdominal pain There may be nausea even vomiting and sometimes unconsciousness Is this the picture that we describe as heart strain? If we pause and analyze the symptoms we find very little evidence that the circulatory system is at fault and we are forced to conclude that the term heart strain is a much misused expression When the term 'strain is used in connection with a skeletal muscle I understand a stretching or tearing of the muscle where it meets its tendon or of the tendon itself I can even visualize the rupture of a few strands of muscle with subsequent soreness and stiffness at the site but this same injury does not occur in the musculature of the heart Nature protects so vital a structure from injury that is so easily produced Moreover such an injury has never been seen postmortem in healthy hearts We do meet rare cases of rupture of the wall of the heart but rupture does not occur unless the area is the site of an infarct resulting from a previous coronary occlusion Authentic cases of spontaneous rupture of the healthy heart have never been reported Barth has collected 24 instances of spontaneous rupture of the heart and in every instance the seat of the lesion was in the left ventricle where the heart wall is usually the thickest So we

conclude that the rupture occurred through a degenerated most likely infarcted area

Likewise rupture of a healthy heart valve occurs rarely if ever and cannot be produced by severe exertion or 'strain' When it does occur the valve has been previously weakened by disease Take a section of healthy aorta with its semilunar valves in place and raise the pressure to the breaking point You will see the slender thin aortic valve leaflets hold fast while the thicker aortic wall will stretch split and then give way If similar experiments are tried upon the healthy mitral and tri cuspid valves the artificially increased pressure leads to stretching and regurgitation through the valve orifice rather than rupture of the tough valvular tissues We may safely conclude from this evidence that severe sudden exertion in the athlete will not produce strain or muscle injury to the heart such as we see in the skeletal muscles nor will it damage or rupture the valvular leaflets

Even the sudden death of an athlete at the finishing line (an extremely rare event) is not necessarily caused by the heart It may conceivably occur from an overlooked persistent thymus or may be brought about by the mechanism of vagal inhibition If the day is excessively warm and the race closely contested the possibility of heat stroke or exhaustion must be kept in mind May there not exist also, under these dramatic cir cumstances a condition closely akin to surgical shock? The psychic strain is certainly tremendous particularly in the much advertised stars before and during a hard race and is it not possible that the nervous system may be at fault and not the cardiovascular? Surgeons are notably heart conscious and in my experience they are inclined to blame on the heart all sudden deaths during or just after operations Here too the effect of the nervous system the ductless glands and other factors pro ducing shock are often overlooked The toxins of infection may leave in their wake not so much direct cardiac disability as a distinct loss of tone in the vasomotor system It is the latter condition that slows down the athlete Earlier exhaustion is noted many times in students when they have been permitted to resume activities too soon after recent infections like influenza

Acute cardiac dilatation is the most popular diagnosis advanced to account for symptoms observed in the athlete at the end of the race Indeed this diagnosis has enjoyed widespread popularity mainly be cause accurate methods of investigation have not been used Reports have been based on the percussion of the hearts of athletes a most unreliable method especially in heavy set well developed men Percussion usually gives a larger cardiac outline than the roentgen ray and this may be the main reason why the heart of the athlete has for many decades been wrongly suspected of showing hypertrophy caused by exercise Ortho diascopy is a better method to determine cardiac size of athletes and when carefully performed is cheaper and better suited to the need of a university health service

Immediate examination of the hearts of athletes with the roentgen ray as they fall exhausted over the finish line shows a heart shadow which is not that of acute cardiac dilatation in most instances the shadow is actually smaller than the normal Richards,[31] and later Gordon,[136] by a series of pictures on marathon runners showed that all hearts are smaller immediately after the race and return to normal size during the course of a day Experimental studies on animals which have been properly carried out and controlled seem to show a smaller heart when the point of exhaustion is reached So we must abandon the term acute cardiac dilatation when referring to the cause of exhaustion in athletes with normal hearts Even without the experimental proof which I have mentioned, the idea should appeal to us as being anatomically unsound The heart is limited in its power to dilate, especially if the process is acute by *the tough fibers of the pericardial sac If we perform Muller's experiment* under the fluoroscope we can see the heart dilate to the limit of its pericardial restraint and assume normal size as soon as the experiment is discontinued if healthy it is none the worse for the experience

The cause of the smaller heart size observed in the athlete after sudden exertion taxes our power of speculation The skeletal muscle, when subjected to overuse shows cramp and stiffness because of diminution in blood supply The overaction with the attending imperfect oxidation gives a swelling of the skeletal muscle fibers with tension of the sheaths Later there develops the painful stiffness so commonly met in those unaccustomed to exercise We cannot conceive of this phenomenon occurring in the healthy heart It is a vital organ necessary for the survival of the organism constantly beating with relatively short rest periods and with a more abundant circulation It is tempting to explain the smaller size of the healthy heart after severe exertion by an excess of lactic acid, but this has not been proved

Lewis [8] has come to the conclusion

> that the burdens imposed by physiological acts upon the normal heart however heavy these burdens may be never injure the heart fibers never produce injurious dilatation and never exhaust the heart's reserve

We must accept this explanation on the basis of experimental proof The heart of the normal athlete is an organ with a reserve far in excess of the organs and systems it supplies It cannot be strained because other bodily mechanisms have lower protective thresholds For example skeletal muscle cramp or cerebral anemia will set in and stop the athlete before the heart gives out Lately more knowledge of the effects of hypoglycemia has led us to attribute to that syndrome the condition of the athlete at the conclusion of the race The blood sugar levels of runners who are forced to drop out of marathon races is supporting evidence of this theory since on numerous occasions the blood sugar has been found almost to approach the level at which unconsciousness comes on

We must therefore take into consideration all these mechanisms before we blame the symptoms of dyspnea rapid pulse exhaustion or even coma on the heart Consider always the fact that Nature surrounds and protects the heart by a series of defensive barriers because it is so indispensable to life

One of the most discussed and interesting questions relating to the problem of the heart in athletics is the so called chronic enlargement supposedly resulting from strain long continued and referred to for years as athlete s heart" If we approach this problem by studying the hearts of lower animals and drawing conclusions from what we observe in them we may easily be misled Clark points out some striking differences in the heart weights in proportion to the body bulk of active and inactive animals and shows that dogs bred for speed have higher heart ratios than ordinary dogs Likewise Fox has shown that quiet birds like the owl have smaller heart ratios than active fliers This author also describes hypertrophy of the heart in captive mammals and birds, but he cautions us that the essential character of these changes is open to dispute in the absence of proof that some unrecognized previous infection or toxemia did not exist Since there are some striking differences in the physics of the circulation in the lower animals particularly in birds we must be on our guard and not draw general conclusions too readily However if work hypertrophy occurs among the commoner beasts of burden we should reasonably expect to find the pathologic museums of our veterinary schools crowded with these specimens Inquiry reveals that this is not the case

Studies by Gordon Richards and my own observations all show quite conclusively that enlargement of the heart of healthy athletes does not occur even when the period of training extends over many years Consequently we do not believe that participation in college athletics predisposes to cardiac enlargement and to the disability which commonly comes to those who have large hearts We cannot deny that the heart muscle develops the same as any other muscle and benefits from exercise which it needs whether healthy or diseased In consequence of this development there is it is true an increase in size but the amount of this increase relative to the initial bulk is small We know that the heart does not enlarge with exercise in the same ratio as skeletal muscle but even though we assume that it does the increase in size as we note it under the fluoroscope falls far short of the enlargement commonly seen as the result of disease We must admit at this stage of our discussion that concentric hypertrophy of the heart may be present and escape recognition under the fluoroscope but this form is rarely in evidence at postmortem

Gordon concludes from his careful study of marathon runners that many years of training and competition do no cause cardiac enlargement Pancoast likewise after examining the chest plates of many athletes found no cardiac enlargement in any of them He observes that among them was one of the world s greatest runners who showed no cardiac enlargement in spite of the fact that one lung was almost obliterated by pleural thick

ening and adhesions The examination of transcontinental foot runners who ran 3434 miles in 84 consecutive days, showed heart diameters within normal limits The only large hearts I have seen in athletes have been those secondary to previous unrecognized rheumatic infection

The relationship between active participation in school and college athletics and the early development of cardiovascular degenerative changes is another problem deserving attention The sudden death in middle life of a man who had previously been a world s record holder will be widely discussed and cited as an example of the ill effects of athletics on the cardiovascular system in later life Hundreds of others who engaged in the same form of training at the same university and who ran in the same races will be forgotten and their records uninvestigated Careful studies of athletes in later life are rare in the literature Morgan in his analysis of the first 24 Oxford and Cambridge boat races from 1829 to 1869 both in clusive in which he obtained information from all of the 255 living members of the crews except four showed that there is little appreciable difference in the mortality from heart disease among university oarsmen of corresponding age Edgecombe[87] also after careful inquiry into the histories of a large number of university oarsmen showed that they are no more short lived than nonathletic men nor are they prone to die of heart affections

The sustained interest in all forms of athletic activity in America should impress upon university officials the duty of providing the equipment necessary to carry out a complete study of the cardiovascular system of all athletes The usual methods of percussion a blood pressure reading and auscultation no longer suffice Fluoroscopy is needed to determine ac curately the cardiac size and shape Likewise routine electrocardiograms are indicated for those students who display any of the arrhythmias or who give a history of antecedent rheumatic infection

A special form for this study may be useful (Table XV) It should include a complete survey of the past medical and family histories We sometimes find the family history of great value when we are called upon to render a decision as to the advisability of a student engaging in athletic competition Should one or more members of the family show evidence of early cardiovascular degenerative changes we are apt to study the student more closely and to note in detail the response to exercise In this group special attention should be paid to any abnormal blood pressure elevations if long sustained The blood vessels of the eye grounds should be examined frequently for early changes

When the cardiovascular examination of each student is completed, the school physician should write a full opinion based on these findings The evidence should be weighed and the decision given and this should be done unhesitantly for uncertainty on the part of the examiner plays havoc with the mental condition of the student

Diseased hearts should be carefully and completely studied These stu dents should not be sent out with the cautiously imparted knowledge that

TABLE XX

FORM FOR STUDY OF ATHLETES

NAME
Address — Age — Date
Height
Weight

HISTORY
Previous general state of health
Has heart disease ever been diagnosed?
(When and by whom)
Colds — Scarlet fever
Tonsillitis — Diphtheria
Tonsils removed — Pneumonia
Chorea — Influenza
Growing pains — Recent loss of weight
Rheumatic fever — Operations
Thyroid disease — Other illnesses
Kidney disease
Athletic history — Sports indulged in
Endurance
Any family history of — Heart disease
Kidney disease
High blood pressure
Tuberculosis
General condition at present — Fatigue — Pa n over heart — Shortness of breath
Palpitation

EXAMINATION
Type of individual — Build — Type of chest
Physique
Thyroid — Blood pressure — Pulse
Heart outline
Auscultatory findings
Electrocard ogram
Orthodiagram
DIAGNOSIS
Recommendations

they have heart trouble and should take it easy They need exercise and careful grading of their exercise They should be studied from the standpoint of the functional capacity of the cardiac muscle We cannot deny the fact that some forms of athletic activity have a definite value in the treatment of cardiovascular disease Furthermore in cardiac cases when compensation is good we may be gratified to observe some decrease in the heart size after participation in the graded exercises designed to fit their tolerance

A prescription of rest in the case of the student in whom the only finding is an apical or pulmonic systolic murmur is indeed radical advice He misses thereby much that college life offers the companionship of fellow athletes the sportsmanship that competition develops and the beneficial effects of sports in building his body for we cannot deny that properly regulated athletic training increases the efficiency of the heart assists the blood and lymph flow increases the vital capacity stimulates metabolic activity develops a higher resistance to bacterial invasion and if well regulated will act as a psychic diversion and improve the tone of the central nervous system If the student is denied these advantages on the

basis of a single incomplete (often only auscultatory) examination of the heart he will grow up with the belief that he is physically inferior Psychiatrists tell us that this may prove a serious blow to character formation The growing boy s whole attitude toward life may be adversely influenced to mention nothing of the possibilities of the effect of the lack of exercise on the tone of the cardiac and skeletal musculature

# 21

## DIET IN HEART DISEASE

I saw a few die of hunger of eating a hundred thousand
—BENJAMIN FRANKLIN *Poor Richard* 1736

Careful regulation of the diet is an important part of the management of the cardiac patient While dietotherapy occupies a foremost place when diabetes complicates cardiac disease proper attention must also be given to this aspect of therapy in the presence of obesity and hyper tension The object of any dietary regime is to lessen the work placed upon the heart and when this is accomplished it will greatly aid the other forms of therapy in maintaining circulatory efficiency

Unfortunately the subject of dietetics is uninteresting to many physi cians and as a result cultists have made extensive inroads in this impor tant field The patient of today is interested in dietary matters and usually turns to the faddist only when he fails to get the necessary information on the subject from his medical adviser The obligation cannot be filled by having at hand printed sheets containing dietary instructions The patient is an individual and likes individual attention Consequently the value of any dietary scheme is greatly increased when it is written out for the patient at the time when treatment is discussed Moreover it is ap preciated is talked about in the hinterlands and as a result is much more apt to be followed Before considering the detailed dietary manage ment of the various types of heart disease the principles upon which these diets are constructed will be reviewed briefly

An adequate diet is made up of proteins fats carbohydrates salts water and vitamins It should have a caloric value sufficient to meet basal metabolic requirements plus the stimulating action of certain foodstuffs and the demands made by the type of work performed by the patient The average requirements for a man doing light work is 3000 calories per day while the average requirement for a woman is 2700 calories

Protein furnishes the material for the growth and repair of the body tissues and is an essential constituent of the diet While the adult does not require protein for growth it is still needed for tissue replacement All proteins consist of giant molecules that are split up during digestion into somewhat over twenty simple compounds that are known as amino acids These substances are further broken up into glucose and fatty acids Protein contains 16 per cent nitrogen, consequently each gram of nitrogen is equivalent to 6 25 Gm of protein metabolized The daily intake of protein should be about 1 Gm for each kilogram of body weight Man is a fortunate animal for in health adaptation is possible on diets that contain

a wide variation in the quantity of protein Inhabitants of the Arctic Circle for example may live indefinitely on a strictly carnivorous diet The cardiac patient should always u e great care in the amount of protein foodstuffs that he selects While an amount sufficient to replace ordinary wear and tear should be allowed, amounts above 50 Gm a day may accelerate metabolism and cardiac rate and in this manner increase the work of the heart Consequently the specific dynamic action of this food stuff is a factor to be reckoned with at all times It is evident that over exertion after a meal rich in protein places a load on the myocardium that often has disastrous results The protein in the diet of the ambulatory cardiac patient should therefore be restricted and the amount may be as low as 0 5 Gm per kilogram of body weight

Carbohydrates are important constituents of the diet of the cardiac patient since they furnish the contracting heart muscle with its most readily assimilated food For this reason carbohydrates should make up well over 50 per cent of the energy content of the diet in patients who suffer from heart disease At the present time this is no burden to the poorer classes since nearly all carbohydrates are cheap and readily obtainable

Fats do not hold a place of importance on the menu of the cardiac patient In large amounts they exert a depressing effect on gastric secretion and slow the emptying of the stomach For this reason they should b used sparingly since all foods recommended to a patient who has advanced heart damage should be easily digested According to many writers, an excess of fat in the diet plays a part in the development of the generalized arteriosclerosis in diabetes nephritis and nephrosis The results of future experiments must be awaited however to prove the exact relationship of excess fat in the diet to degenerative changes in the pre ence of a normal metabolism

The mineral constituents of the diet are also important to patients who suffer from cardiovascular disease and may be obtained in the required amounts in milk and vegetables Sodium chloride is perhaps the only salt consumed as such and the regulation of its daily intake warrant consideration in the presence of congestive failure About 1 to 2 Gm of sodium chloride are needed daily but the average person consumes amounts far in excess of this figure

Calcium is essential, especially in children and unless the diet is carefully planned it is much more likely to show a deficiency of this element About 0 9 to 1 0 Gm of calcium is required daily and this can best be obtained in milk Approximately 12 mg of iron constitute the daily requirement although in pregnancy this figure is increased Iodine is essential to prevent the development of simple goiter and should be given in districts where there is a known deficiency This requirement may be met by the use of iodized salt

Patients on adequate diets usually obtain a sufficient supply of vitamins from the ordinary foods The green vegetables and fresh fruit juices supply

vitamin C, whole wheat bread and the preparations containing wheat germ furnish vitamin B whole milk meat fat and eggs add a plentiful supply of vitamins A and D The latter can be increased by the addition of cod liver oil The relationship between the vitamins and circulatory efficiency will be taken up in greater detail later (page 552)

## ANEMIA

Mild grades of secondary anemia have no demonstrable effect on the heart Severe secondary anemias and primary or Addison s anemia may produce fatty degenerative changes in the cardiac muscle High grades of anemia may contribute to the production of congestive failure in rare instances and anginal symptoms may arise from this alteration in the quality of the blood reaching the myocardium (page 423)

The heart disabled by anemia responds to liver therapy and a diet rich in substances that contain the anti anemic factors e g liver or its equivalent vitamins and soluble iron salts In anemia when achlorhydria is present gastrointestinal symptoms that are often productive of secondary cardiovascular effects may be prevented by suitable amounts of dilute hydrochloric acid taken with the meals

Weakness palpitation and tachycardia are other cardiac symptoms that often appear in patients who have severe anemias of the secondary type These may disappear when the cause of the anemia is discovered and treated

## ARTERIOSCLEROSIS

The relationship between diet and arteriosclerosis has been a favorite problem for investigators for many years Diets containing the various foodstuffs in different proportions as well as in excessive and insufficient amounts have been fed to laboratory animals Patients with disorders of metabolism have also been studied for some possible clue regarding the relationship of substances in the diet to the speeding up of the process of sclerosis

The results of animal experimentations have been interesting and suggestive but wide conclusions should not be drawn The feeding of meat to rabbits has been shown to result in considerable aortic sclerosis while rabbits on milk and egg diets develop extensive intimal sclerosis Cholesterol has the same effect according to some observers Others claim that the high protein diet causes the arterial changes that uniformly appear in these laboratory animals

Coronary sclerosis has also been noted in white mice following diets high in cholesterol Similar changes however have not been seen in dogs and other carnivorous animals We may surmise that the abnormal substances in the diets of the rabbits and mice produce deviations in metabolism and perhaps account for the arterial changes In any event we are driven

back to cholesterol metabolism and must admit that it differs in various species under the same conditions

In man we cannot say that excess intake of foods gives arteriosclerosi If obesity is directly related to hypertension (which I doubt), then it may influence in this manner the state of the arteries Undernutrition apparently does not lead to arteriosclerosis for if it did, this lesion would be a preva lent disease in many countries of the world today If high protein diets re sult eventually in arterial changes studies of Eskimos should show a marked evidence of arteriosclerosis This is not the case On the other hand races that exist mainly on carbohydrates exhibit abundant sclerosis so we can not blame the high protein diets for degenerative arterial changes

Joslin s studies[176] seem to show that in diabetes the excess fat in the diet contributes to early sclerosis but here we must remember the metabolism is decidedly abnormal Neither can we blame the development of arteriosclerosis on diets that consist largely of alcoholic beverages for we do not find advanced changes in the vessels of alcoholics when viewed in the light of the age of the patient

It is a common observation that patients who have widespread arterio-sclerosis accompanied by advanced changes in the coronary tree gradually lose weight This has been referred to as cardiac cachexia In some cases it may be accelerated by the effects of congestive failure on the digestive tract but it is seen also in patients who never show congestive phenomena Moreover, in the latter group, a marked loss of weight may occur when the appetite is good and when the intake of food is sufficient to meet the bodily requirements Beyond a doubt the arteriosclerotic changes inter fere in some way with the proper absorption and utilization of foodstuffs These patients therefore, should be fed a rich full diet Large meals should not be allowed since the same result is obtainable through frequent feedings that have a small bulk but a relatively high food value

In this modern age we continually strive to make our treatment con form to the most recent opinions emanating from research laboratories The family physician of the old school in treating many members of the same family called the arteriosclerosis that developed at an early age in successive generations a diathesis If pressed in the matter of definition he would tell you "It s bred in them In these patients we should plan treatment not entirely along dietary lines but more toward the preven tion of infection and the better regulation of damaging occupations if we hope to avoid early arterial changes The diet should of course be regu lated in the presence of diabetes and obesity

In old age with the usual degree of arteriosclerotic changes present a slight weight loss of ten pounds or so should not in itself be cause for alarm While it has been truthfully said that the worst thing that can happen to an old man is to acquire a young wife who is a good cook most of the blame for the abnormal appetites of old people lies in the condition of their gastro-intestinal tracts possibly associated with un balanced diets Abnormal sensations of hunger and capricious appetites

are common and are often associated with atrophic changes in the stomach and the secreting glands as well as atrophy of the usual reflex pathways While a diet complete in all the foodstuffs should be given to the older patient if there is demonstrable cardiac damage the amount of the food and the speed of its intake should be carefully guarded Circulatory accidents in old people many times can be avoided if these principles are followed

## HYPERTENSION

Diet faddists draw many followers from this large group and in consequence much nonsense continues to be preached by radio programs and magazine articles regarding the cure of hypertension by dietary measures The main darts are directed toward the proteins (the red meats of the trolley car conversations) and salt although fats and carbohydrates are not neglected

Protein restriction has enjoyed a long popularity It has appeal and in the American home where meat appears in such abundance it may have some value In patients with hypertension who show good renal function, no nitrogen retention and slight if any cardiac damage great restriction of proteins is certainly not indicated However so deeply do press and radio influence the American public that to make a statement of this nature to a patient of the hypertensive type trained in the present belief is usually sufficient to undermine confidence at once The hypertensive tourist of physicians offices will usually co-operate in the matter of drug therapy (so many times not indicated) and may even agree to a major operative procedure yet he cannot be induced to eat meat

Experimental evidence fails to show that excess of protein tends to elevate the arterial pressure On the other hand a low protein diet long continued may lower the blood pressure but this is accomplished through the anemia and physical weakness that are produced Both of these undesirable end products of faulty dieting may contribute to an earlier cardiac breakdown Cardiac cases following a long term in one of the dietary camps often show marked improvement when induced to take protein Even in the presence of a complicating renal lesion particularly of the nephrotic type with considerable albuminuria the addition of protein to the diet is an important factor in clearing the edema Consequently in the absence of cardiac failure proteins should be retained in the diets of patients suffering from essential hypertension in the amounts recommended for the normal person

Salt restriction is another popular practice with deep roots in the small volumes written to advise the laity in matters medical Extensive investigations again fail to show that rigid salt restriction has any effect on the clinical course of hypertension Neither will excessive quantities elevate the blood pressure Those who have tried living on salt poor diets can attest to the fact that they never look forward to meal time and many ill advised

cardiac patients do poorly because of the anorexia and loss of strength that follow salt free diets A normal intake of sodium chloride should be permitted in the absence of congestive failure During congestive failure patients should be warned against excess quantities of salts and salty foods Seasonings of all kinds should likewise be prohibited Recently the salt in the diet has become a most important consideration when organic mercurial diuretics are employed over long periods (page 90)

If obesity accompanies hypertension the diet is written accordingly otherwise a normal diet should be allowed The patient must be cautioned against overeating and too rapid eating particularly when the anginal syndrome accompanies hypertension Fluids need not be restricted in the absence of congestive failure although too much fluid should not be allowed

## OBESITY

Obesity is a well recognized handicap to the patient who has cardiac disease While the extra weight in itself is a burden to the heart the increase in the size of the surface area in these cases places an additional load on the peripheral circulation The extra fat accumulating about the heart may also interfere with its free contraction and infiltrate the organ with further impairment in function

Treatment of the obese cardiac patient by dietary measures lessens the work of the heart by lowering the basal metabolism Since protein foods furnish the greatest stimulus to metabolism care should be taken to reduce the intake of this constituent of the diet to the lowest possible figure usually 1 Gm or less per kilogram of ideal or predicted weight

Reduction of the diet should be brought about gradually The caloric need of the patient should always be calculated and a diet prescribed that is below this basic level It is well to proceed slowly by choosing a diet at first that contains a fairly high allowance Carbohydrates should be given to the extent of 0 6 Gm for every gram of protein to maintain nitrogen balance for carbohydrate in this proportion acts in the capacity of protein sparer Very little fat is allowed in the diet, and the body is encouraged to use the fat in its depots to make up for the deficient caloric intake Green vegetables supply vitamins and their bulk satisfies hunger Between meals especially at the start of the regime occasional glucose candies may be allowed to provide quickly utilizable energy and to combat exhaustion

If the ambulatory cardiac patient who is overweight can be encouraged to co-operate and lose on the average of six pounds in one month much improvement in the subjective symptoms will follow Diets calculated to provide 1000 to 1200 calories are employed in the majority of cases

In patients who require only mild reduction programs, the qualitative restriction shown in Table XVI* may be sufficient For the convenience of

* Tables XVI to XXIII reprinted from Treatment by Diet by Barborka J B Lippincott Company

TABLE XVI

SIMPLE QUALITATIVE RESTRICTION

TYPICAL FOODS ALLOWED IN REDUCTION DIETS

Milk skimmed and buttermilk
Egg
Meat lean
Meats low in fat as
  Chicken—lean portion
  Liver
  Fish other than salmon
  Shrimp
  Crabmeat
Fruit 5 and 10%
Vegetables 3 and 6 %
Clear soup

TYPICAL FOODS TO AVOID IN REDUCTION DIETS

Sugar and All Sweets
Starches as
  Bread
  *Cereals*
  Macaroni
  Spaghetti
  Pastry pie cakes
  Sweet desserts
Vegetables High in Carbohydrates as
  Potato
  Shelled peas
  Shelled beans
  Corn
  *Parsnips*
Fats as
  Butter
  Salad oils
  Cream
Meats high in fat s
  Pork
  Lamb chops with large amounts of fat

*the reader* a classification of the fresh fruits and vegetables according to their percentage of carbohydrate content has been included (Table XVII) A few sample reduction diets suggesting the amount and distribution of the daily food allowance that may serve as patterns when writing these dietary prescriptions will be found in Tables XVIII XIX XX

## RHEUMATIC HEART DISEASE

The dietary regime in patients suffering from active infection should be planned to combat the wasting effect of the long febrile siege All the food that can be comfortably and safely taken should be allowed and in this regime carbohydrates predominate since every effort is made to keep up body weight Vitamin C has been claimed to have a direct relationship to rheumatic infection and has been given in large amounts in the diets of these patients but I have never seen any marked benefit follow its administration Fluids should be given freely owing to excessive sweating and

TABLE XVII

CLASSIFICATION OF FRESH VEGETABLES AND FRESH FRUITS ACCORDING TO PERCENTAGE OF CARBOHYDRATE CONTENT

VEGETABLES

| 3% | 6% | 15% | 20% |
|---|---|---|---|
| Asparagus | Artichoke French | Parsnips | Corn |
| Beet Greens | Beet | Salsify | Garlic |
| Broccoli | Carrot | Pea | Horseradish Root |
| Brussel Sprouts | Celeriac | | Potato |
| Cabbage | Dandelion Greens | | Dried Bean |
| Chinese Cabbage | Kale | | |
| Cauliflower | Kohl Rabi | | |
| Celery | Leeks | | |
| Cucumber | Onion | | |
| Eggplant | Parsley | | |
| Endive | Pea (tiny) | | |
| Lettuce | Pumpkin | | |
| Marrow | Rutabaga | | |
| Mustard Greens | String Bean (mature) | | |
| Green Pepper | Squash | | |
| Okra | Turnip | | |
| Radish | | | |
| Sauerkraut | | | |
| Sorrel | | | |
| Spinach | | | |
| String Bean (tiny) | | | |
| Summer Squash | | | |
| Tomato | | | |
| Watercress | | | |

FRUITS

| 5% | 10% | 15% | 20% |
|---|---|---|---|
| Muskmelon | Blackberry | Apple | Banana |
| Honeydew | Cranberry | Apricot | Fig (fresh) |
| Watermelon | Gooseberry | Blueberry | Grape Juice |
| Rhubarb | Grapefruit | Cherry | Fresh Prune |
| *Avocado | Lemon | Currant | |
| | Lime | Grape | |
| | Orange | Guava | |
| | Papaya | Huckleberry | |
| | Peach | Nectarine | |
| | Pineapple | Papaw | |
| | Strawberry | Pear | |
| | Tangerine | Plum | |
| | | Quince | |
| | | Raspberry | |

* Avocados contain 17 2 per cent fat

an increase in the sodium chloride intake is good therapy for the same reason

## CARDIOVASCULAR SYPHILIS

The drastic cures for aneurysms recommended by early writers included strenuous dietary measures as well as rest and venesection Fluid and food were reduced to extremely low levels in the regime originally suggested by Albertini and Valsalva in the seventeenth century (page 252) Today no particular dietary program for cardiovascular syphilis is recom

# DIET IN HEART DISEASE

## Table XVIII
### REDUCTION DIET
FOR EXPECTED OR IDEAL WEIGHT OF 160 POUNDS

**800 Calories**

*Suggested Distribution of the Total Food Allowance for One Day*

| | | Grams |
|---|---|---|
| **BREAKFAST** | | |
| Fruit 10% | 1 serving | 100 |
| Egg | 1 | 50 |
| Egg whites | 2 | 66 |
| Bread | ½ thin slice | 10 |
| Butter | ¼ square | 3 |
| Beverage—coffee or tea | | |
| **LUNCHEON** | | |
| Meat (low in fat) | 1 large serving | 90 |
| Vegetables 3% | 2 small servings | 150 |
| Fruit 5% | 1 serving | 100 |
| Milk (skimmed) | 1 glass | 200 |
| **DINNER** | | |
| Meat (lean) | 1 large serving | 90 |
| Vegetable 6% | 1 small serving | 75 |
| Salad vegetable 3% | 1 small serving | 75 |
| Fruit 10% | 1 serving | 100 |
| Milk (skimmed) | 1 glass | 200 |

## Table XIX
### REDUCTION DIET
FOR EXPECTED OR IDEAL WEIGHT OF 160 POUNDS

**1 000 Calories**

*Suggested Distribution of the Total Food Allowance for One Day*

| | | Grams |
|---|---|---|
| **BREAKFAST** | | |
| Fruit 10% | 1 serving | 100 |
| Bacon | 1 slice crisp | 5 |
| Egg | 1 | 50 |
| Bread | ½ thin slice | 10 |
| Butter | ½ square | 5 |
| Beverage—coffee or tea | | |
| **LUNCHEON** | | |
| Meat | 1 large serving | 90 |
| Vegetables 3% | 2 small servings | 150 |
| Butter | ½ square | 5 |
| Fruit 10% | 1 serving | 100 |
| Milk (skimmed) | 1 glass | 200 |
| **DINNER** | | |
| Meat | 1 large serving | 90 |
| Vegetable 6% | 1 small serving | 75 |
| Salad vegetable 3% | 1 small serving | 75 |
| Butter | ½ square | 5 |
| Fruit 5% | 1 serving | 100 |
| Milk (skimmed) | 1 glass | 200 |

TABLE XX

REDUCTION DIET

FOR EXPECTED OR IDEAL WEIGHT OF 160 POUNDS

1 200 Calories

*Suggested Distribution of the Total Food Allowance for One Day*

| | | Grams |
|---|---|---|
| BREAKFAST | | |
| Fruit 10% | 1 serving | 100 |
| Bacon | 1 slice crisp | 5 |
| Egg | 1 | 50 |
| Bread | ½ thin slice | 10 |
| Butter | 1 square | 10 |
| Cream 20% | 1 tablespoon | 15 |
| Beverage—coffee or tea | | |
| LUNCHEON | | |
| Meat | 1 large serving | 90 |
| Vegetable 3% | 2 small servings | 150 |
| Butter | 1½ squares | 15 |
| Fruit 10% | 1 serving | 100 |
| Milk (skimmed) | 1 glass | 200 |
| DINNER | | |
| Meat | 1 large serving | 90 |
| Vegetable 6% | 1 small serving | 75 |
| Salad vegetable 3% | 1 small serving | 75 |
| Butter | 1½ squares | 15 |
| Fruit 5% | 1 serving | 100 |
| Milk (skimmed) | 1 glass | 200 |

mended The content of the diet prescription is entirely governed by the patient s weight and the presence or absence of congestive cardiac failure We must admit however in the light of modern discoveries of the physiologic chemists, that the old starvation treatment of Albertini and Valsalva was not without its effect on the basal metabolism blood pressure and pulse rate and certainly must have produced in this manner the favorable (although temporary) effect they claimed for it

## DIET IN CONGESTIVE FAILURE

With the onset of congestive failure diet is a most essential part of the management When the patient is first seen the presence of dyspnea and alimentary tract congestion generally places the thought of food in the background

At the start the fluid intake should be restricted It is a custom to begin treatment of congestive failure with the diet originally proposed in 1866 by Karell a Russian court physician While not originally prescribed for patients with this type of heart disease but rather for 'les hydropsies de toute natur ' Karell s diet consisted of skimmed milk divided into four feedings of 200 cc each given at 8 A M 12 M 4 P M and 8 P M This regime furnishes 26 Gm of protein 1 6 Gm of NaCl 800 cc of fluid and approximately 550 calories It may be continued for one or two days Pa

tients who cannot take milk may substitute other articles to make up a total of the same caloric value from the following list orange juice cereal gruel (Pablum*) thin cream soups or buttered toast As a rule it is well to restrict the fluid intake to 1200 cc during the first 24 hours When satis

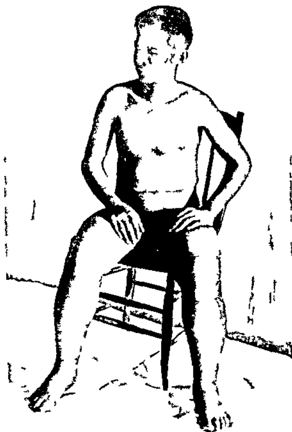


Fig 163 Beriberi (Wet type) (Courtesy Philippine Bureau of Science)

factory diuresis has been obtained the fluid intake may be increased to an extent compatible with comfort and the total urinary output

When improvement in the patient's condition occurs little further attention is usually given to diet However the amount and type of food ingested is most important at this stage and greatly influences the cardiac mechanism through its effect on the basal metabolism Undernutrition in normal

* Mead Johnson Company

individuals has been shown to be accompanied by a fall in the pulse rate blood pressure and metabolic rate These alterations follow a slight weight loss and appear even though the usual activities are unrestricted If we produce similar physiologic alterations during convalescence from congestive failure we shall greatly aid cardiac recovery Consequently restriction of the diet for the first two or three weeks of the patient's convalescence is helpful Proger[307] advises first a severe and then a more moderate dietary restriction over this period, so that about 10 per cent of the patient's body weight is lost In three weeks the patient should be raised to a maintenance diet During this entire period, fluids may be restricted, for a drop in the level of fluid exchange causes the patient to drink less

This temporary regime of undernutrition produces in the cardiac patient who is convalescing from an attack of congestive failure a further slowing of the pulse beyond the degree obtained by bed rest and digitalis and an additional increase in blood pressure The cardiac output is less and the condition of the heart improves because it has less work to do at the lower metabolic level In addition as weight is lost, the vital capacity increases Measurements in some cases may show a decrease in cardiac size When the same dietary principles are employed in ambulatory cardiac patients, an improvement in the exercise tolerance is noted During the first days of the dietary restriction hunger and weakness may be prominent subjective complaints The co-operative patient is usually satisfied with the diet when its purpose is explained especially if the improvement is evident following the loss of weight

If the patient has had several attacks of congestive failure and considerable weight has already been lost, the period of undernutrition should be omitted and the diet kept above maintenance levels Obese patients many times do not experience the beneficial effects above described The best results may be obtained in cases where the patient is normal in weight or slightly above the normal, although individual variations in this response may occur

TABLE XXI

MODIFIED KARELL DIET

| | | Grams |
|---|---|---|
| | BREAKFAST | |
| Bread (toast) | ½ thin slice | 10 |
| Fruit 10% | 1 serving | 100 |
| Sugar | 1 teaspoon | 5 |
| Egg | 1 | 50 |
| | LUNCHEON | |
| Bread (toast) | ½ thin slice | 10 |
| Cream soup | ⅔ cup | 150 |
| Butter | ½ square | 5 |
| | DINNER | |
| Bread (toast) | ½ thin slice | 10 |
| Milk | 1 glass | 200 |
| Butter | 1 square | 10 |
| Egg | 1 | 50 |

The diets shown in Tables XXI, XXII, XXIII may be used as patterns when variations of the Karell diet or increase in the caloric intake are called for following an attack of congestive failure.

TABLE XXII
HEART DISEASE
CARDIAC DIET I
45 Grams Protein
1400 Calories

*Suggested Distribution of the Total Food Allowance for One Day*

| | | Grams |
|---|---|---|
| **BREAKFAST** | | |
| Fruit Juice 10% | ¼ glass | 50 |
| Cereal (cooked) | ½ cup | 105 |
| Egg | 1 | 50 |
| Bread (toast) | 1 thin slice | 20 |
| Butter | ½ square | 5 |
| Cream 20% | ¼ cup | 60 |
| Beverage—coffee substitute | | |
| 10:00 A.M. Fruit juice | ½ glass | 100 |
| **LUNCHEON** | | |
| Soup | | |
| Vegetable puree 3% or 6% | ½ cup | 100 |
| Milk | ½ glass | 100 |
| Butter | ½ square | 5 |
| Egg or egg substitute | 1 | 50 |
| Bread (toast) | 1 thin slice | 20 |
| Butter | ½ square | 5 |
| Fruit puree 10% or 15% | ½ cup | 100 |
| 3:00 P.M. Milk | 1 glass | 200 |
| **DINNER** | | |
| Fruit juice 10% | ¼ glass | 50 |
| Milk toast | | |
| Milk | 1 glass | 200 |
| Toast | 1 thin slice | 20 |
| Butter | ½ square | 5 |
| Custard | ½ cup | 100 |
| Bedtime Fruit juice | ½ glass | 100 |

The principles that guide the physician in his selection of a suitable dietary regime for the patient convalescing from an attack of congestive failure may be briefly summarized as follows: add to the intake gradually, always keeping below basal requirements. This may be done by giving frequent small feedings, thus avoiding the strain that large meals place on the cardiac reserve. The cardiac muscle requires an abundance of carbohydrates (glucose) for its recovery, and this constituent of the diet should be proportionately increased. While moderate restriction in fluid intake is essential as long as edema can be demonstrated clinically, it is most important during this period to keep the salt intake low. However, if large amounts of mercurial diuretics are being given, care should be taken not

TABLE XXIII

HEART DISEASE

CARDIAC DIET II

50 Grams Protein

1800 Calories

*Suggested Distribution of Total Food Allowance for One Day*

BREAKFAST

| | | Grams |
|---|---|---|
| Fruit 10% | 1 serving | 100 |
| Cereal (cooked) | 2/3 cup | 140 |
| Egg | 1 | 50 |
| *Bread (toast)* | *1 thin slice* | 20 |
| Butter | 1 square | 10 |
| Sugar | 1 tablespoon | 15 |
| Jam | 1 tablespoon | 15 |
| Cream 20% | 1/4 cup | 60 |
| Beverage—coffee substitute | | |
| **LUNCHEON** | | |
| Meat | 1 small serving | 45 |
| Potato or substitute | 1 small serving | 75 |
| Vegetable 3% or 6% | 2 servings | 200 |
| Bread | 1 thin slice | 20 |
| Butter | 1½ squares | 15 |
| Dessert | 1 serving | 100 |
| Milk | 1 glass | 200 |
| 3 00 P M Fruit 10% or 15% | 1 serving | 100 |
| ***DINNER*** | | |
| Potato or substitute | 1 small serving | 75 |
| Vegetable 3% or 6% | 1 serving | 100 |
| Bread | 1 thin slice | 20 |
| Butter | 1 square | 10 |
| Fruit 10% or 15% | 1 serving | 100 |
| Milk | 1 glass | 200 |

to restrict the intake of sodium chloride too drastically for copious diuresis may give rise to symptoms associated with hypochloremia Protein foods should be given sparingly owing to their specific dynamic action on metabolism Fats should be decreased to a minimum because of difficulty in their assimilation in the presence of the slowing of all the digestive processes

## *THE HEART IN DEFICIENCY DISEASES*

Feeding experiments carried out on laboratory animals have shown that a variety of symptoms may be produced by diets that are lacking in one or more of the vitamins Similar symptoms have been observed in man when diets deficient in these substances have been taken over long periods

Rarely is the clinical picture so clear cut and definite that the exact deficiency is immediately recognized Usually the patient is found to be suffering from a combination of several deficiencies If we consider the effect of vitamin deficiency on the function of the circulation our attention will be focused mainly on vitamin $B_1$ for in the light of recent studies this is the

chief vitamin deficiency associated with major disturbances of the cardiovascular system

Isolation of vitamin B followed investigations of the cause of beriberi This substance was therefore known as the antiberiberi vitamin Still later discoveries proved that vitamin B was a complex substance made up of two or more factors differentiated on the basis of sensitivity to heat The antiberiberi vitamin was designated $B_1$ and the newly isolated substance as vitamin B or antidermatitis vitamin or pellagra preventing (P P) factor Substances like wheat germ yeast whole meal cereals nuts egg yolk liver heart and kidney are rich in vitamin $B_1$ However it may be said that substances rich in one vitamin of this complex also contain varying amounts of the others

In reviewing the circulatory abnormalities that follow a diet deficient in the $B_1$ factor let us consider first the effect of a marked deficiency This results in the disease picture known as beriberi that is commonly encountered in many of the rice eating countries of the East China Japan India the Philippines and the Dutch Indies and less frequently in other countries Beriberi can occur in mild or severe forms causing partial or complete deficiency states at any age or in any race where poverty and ignorance prevail It is frequently associated with a diet of refined cereals for in the milling process the germ of the grain containing the vitamin is removed

In many ways beriberi is a curious disease Variable factors such as age locality type of work and nationality appear greatly to influence the clinical picture and this is particularly true in respect to the cardiovascular system In the infant beriberi usually runs an acute course There may be sudden diminution of urine rigidity of the body dyspnea and cyanosis Weakness rapid pulse and edema of the legs may develop quickly and in the absence of prompt treatment sudden death may occur In the adult the picture is quite different The onset is insidious with vague and general symptoms of fatigue indigestion mild grades of dyspnea tachycardia and tenderness over the muscles Later symptoms pointing to the involvement of the nervous system may develop and degenerative changes appear in the peripheral nerves When neuritis predominates the condition is known as the dry type of beriberi In contrast to this course another patient may show a predominance of cardiovascular symptoms dyspnea marked cardiac enlargement with dilatation and fluid in the serous cavities This is the so-called wet type (Fig 163) Some observers claim that this form is more apt to develop in young adult males engaged in strenuous occupations where neuritis does not appear early and force the patient to rest Gastro intestinal symptoms such as anorexia diarrhea and vomiting may occur in both types

Wenckebach studying a number of advanced cases in Java with Aalsmeer[389] attempted to correlate the clinical and autopsy findings of beriberi and described the cardiac enlargement and edema He popularized the term beriberi heart Wenckebach likewise called attention to the fact that

the active muscles appear to suffer most in this disease: the soleus, the masseter, the gastrocnemius, and finally the heart.

Lack of vitamin $B_1$ causes the heart to lose its power of vigorous contraction, its tone decreases and this is soon followed by dilatation. If we examine the changes that take place in the cardiac tissue and compare them with those observed in the nervous system, we shall find that they are quite similar. Sections of cardiac muscle in beriberi show swelling of the cells caused by retention of water, a condition that differs from ordinary edema. At autopsy, Wenckebach's cases showed dilatation of the right side of the heart: the right ventricle, the right auricle and the conus arteriosus were seen literally to be "blown up" because of the high pressure on the venous side prior to death.

If we view the clinical symptoms in the light of these interesting and curious autopsy findings, the state of the circulation in beriberi becomes evident. At the onset there is only slight edema; all forms of exertion are poorly tolerated, but if they are continued, edema increases and pulsation will be noted in all peripheral vessels. Rest is immediately beneficial in these cases, whereas adrenalin exerts a particularly unfavorable influence. When this drug is injected even in small doses, the diastolic pressure falls promptly to zero, and a pistol-shot sound appears in the femoral vessels. After vitamin $B_1$ is administered in curative doses in advanced beriberi, this reaction to adrenalin disappears in most instances.

These observations suggest that in the wet type of beriberi all the arterioles are widened. The blood literally runs from the arteries into the capillaries and veins. Consequently the systolic energy of the heart is not spent in the capillary bed and the blood pours into the right heart behind a considerably increased venous pressure. Wenckebach described in these cases a "venous hum" in the neighborhood of the crural vein, produced by the increased velocity of the venous blood. The sudden high venous tide causes overloading of a right heart that is already seriously affected by the vitamin $B_1$ deficiency. This explains the "blown up" appearance found at autopsy.

Studies carried out in this country recently have shown some aspects that correspond exactly to these views. A normal or increased velocity of the blood flow with a low arteriovenous oxygen difference have been consistently observed. This is in keeping with the impression that there is present in the wet type of beriberi a general arterial dilatation. The circulation time is an important point in the differential diagnosis between the wet type of beriberi and congestive heart failure. Improvement of the patient following administration of vitamin $B_1$ in beriberi is attended by an increase in the circulation time.

Posterior lobe of the pituitary (pitressin) has a striking effect on the circulatory disturbances of beriberi. Following the injection of this substance the rapid blood flow is checked on the arterial side and the patient notices immediate subjective improvement. The heart rate is slowed, the diastolic pressure increases, the venous pressure falls, the venous hum

disappears and the pistol shot sound is no longer heard In other words the circulation time is slowed and the blood is held longer on the arterial side This of course has raised the question Is the circulatory picture of beriberi due to a decrease of production of the hormone from the posterior lobe of the pituitary gland?

Undoubtedly since the depression years many cases which show cardio vascular symptoms following marked vitamin $B_1$ deficiency have occurred in this country Scott and Hermann[336] observed cases among the rice workers of Louisiana Riesman and Davidson[315] have also reported cases and an instance in a diabetic is recorded by Wohl[419] Nearly every physician will occasionally meet patients who show signs of congestive failure in the absence of a valvular defect or increase of blood pressure or other obvious cause *In all of these instances the nutritional history should be carefully* elicited The patient may be a mixed type and give signs or symptoms of a deficiency state other than cardiovascular for example neuritis or a dermatitis of the pellagra type Various gastro intestinal symptoms may appear If the diagnosis is correct all symptoms should improve following *the administration of vitamin $B_1$*

As we would expect the electrocardiogram reflects the changes that take place in the circulatory system Lately a variety of alterations have been described in the literature Most cases show a sinus tachycardia with a tendency to T wave alterations Some of the T wave alterations that have been encountered in deficiency states suggest coronary occlusion so it is most important to review the clinical findings in all cases which show electrocardiograms of this type In the deficiency states the waves are apt to change more quickly following treatment than they are usually observed to do in the presence of coronary disease Consequently the dis appearance of electrocardiographic deformities following the administra tion of vitamin $B_1$ is a valuable point in differential diagnosis

Weiss and Wilkins[396] described four patients who experienced attacks of syncope in addition to other cardiac manifestations of the deficiency state These patients were found to have a hyperactive carotid sinus reflex (page 379) the slightest stimulation producing asystole and syncope Administration of vitamin $B_1$ abolished the hyperexcitability at once in three of these patients

Considering the number of vitamin $B_1$ preparations now on the market it is interesting to inquire into the effect of vitamin $B_1$ on the normal heart Experiments to determine this have been carried out and very large doses administered to control patients have been found to have no effect on the pulse rate blood pressure electrocardiogram velocity of the blood flow or serum protein and no untoward symptoms have been produced

In many of the cases published in America which show marked cardio vascular symptoms accompanying deficiency of vitamin $B_1$, alcohol has played a major role Alcohol per se is incapable of causing any effect on the heart of laboratory animal or of man However by reason of the large consumption of alcohol diets deficient in vitamin $B_1$ have been taken

by these patients over long periods Today both the neuritis common in alcoholism and the cardiac changes present at times in the same cases are believed to be caused by the vitamin $B_1$ deficiency However, many have shown a polyneuritis before the circulatory changes entered the picture The diets of many of these patients will be found to average well over 4000 calories consequently they may appear to be well nourished in the presence of a grave deficiency

It is important to remember that patients suffering from any type of cardiac disease may have a superimposed deficiency state and congestive failure may be precipitated by this condition Vitamin $B_1$ deficiency should be suspected, particularly in alcoholic patients when the cardiac findings fail to explain the signs and symptoms A therapeutic test of the administration of the vitamin is in order

## ILLUSTRATIVE CASES

The following histories are typical and illustrate types of $B_1$ deficiency that are occasionally met In the first case the diagnosis was not made or suspected The second patient examined at a later date was recognized and proper treatment resulted in rapid improvement

**Case 108** T M age 68 a retired real estate operator lived on a quart of whiskey a day for two months prior to examination (1934) He complained of cough progressive dyspnea and edema

PHYSICAL EXAMINATION showed a normal blood pressure a pulse of 100 with frequent premature contractions marked edema of face and legs and a questionable ascites The heart was moderately enlarged to percussion in all diameters and a blowing systolic murmur was present over the mitral area Urine complete blood count and blood Wassermann reaction were negative

COURSE The patient was placed at bed rest and given digitalis and diuretics which had no effect on his circulatory symptoms His supply of whiskey was difficult to control Removal to a seashore rest home elimination of the whiskey and a normal diet quickly restored his balance The type of circulatory failure was unexplained at this time Follow up examination of the patient (1937) revealed a normal cardiovascular system His wife volunteered the statement that his symptoms recurred in their entirety the previous year when he started drinking again and not eating for three months A normal diet again efficiently removed all signs of congestive failure

**Case 109** J C age 39 a salesman was treated for some years for peptic ulcer with the usual dietary restrictions Gradual loss of weight resulted He was first seen following a vacation during which he consumed considerable quantities of alcohol He took very little solid food during the course of a month He was unable to return to work at the end of this time because of palpitation tachycardia swelling of the hands face and feet and marked dyspnea Previously his exercise tolerance had always been excellent and the history was negative for cardiac disease

PHYSICAL EXAMINATION The blood pressure was found to be low the heart slightly enlarged, and a systolic murmur was present over the aortic area Blood count blood Wassermann reaction and urine were negative

COURSE Hypodermic injections of 30 milligrams daily of thiamin chloride with a diet rich in vitamin B brought marked improvement The patient returned to work in two weeks Subsequent roentgen ray examination revealed a duodenal ulcer necessitating a further adjustment of his dietary regime

## RICKETS AND SCURVY

Rickets may cause cardiac dilatation and failure In children who die suddenly from this disease ventricular dilatation is commonly found at autopsy As a rule rickets is not dangerous to life but deformities may be lasting and when they occur in the chest they may be the cause of alterations in the position and function of the heart

In scurvy cardiovascular symptoms may arise secondary to the anemia not uncommon in this disease or hemorrhage into the pericardial sac Death in some cases may be caused by cardiac failure However Weiss and Wilkins analyzing a control group of 110 cases of scurvy uncomplicated by other types of vitamin deficiency failed to find a single case exhibiting the circulatory abnormalities described here as suggestive of deficiency disease[386]

# 22

# THE SENILE HEART

Sleep after toil port after stormy seas,
Ease after war death after life does greatly please
—SPENSER, *The Faerie Queene* Bk 1 Canto ix, st. 40

Geriatrics deals with the problems of senescence and senility, and usually fails to attract physicians who are searching for special fields of research While questions relating to infancy and childhood are completely studied by well equipped hospital groups and described in separate journals those connected with the sunset of life receive much less attention However there are many therapeutic items of importance in geriatric practice that deserve special consideration Most of them are covered in the limited number of good treatises on the subject of geriatrics, and all are admirably summarized in a recent contribution by Pepper[794] Only problems relating to the cardiovascular system will be briefly considered here

## TYPES

The heart does not share the tendency to atrophy that is present in many other organs of the body as age advances its relation to body weight remains the same Older writers held the opposite view and believed that the heart and arteries underwent hypertrophy Even among physician of today there is a prevailing opinion that continued increase in the blood pressure takes place with advancing years On the contrary, it has been found that the pressures show only slight elevation until the sixth decade and then remain unchanged in the absence of disease the average pressure readings staying at 130 to 140 systolic and 80 to 90 diastolic There is a slight decrease in the pulse rate in the later decades

The change that may be clearly visualized in the senile aorta at the time of fluoroscopy is generally caused by a deposition of calcium in the media Similar alterations occur in the peripheral arteries and are associated with little interference in the passage of blood This condition of the media is known as Monckeberg s sclerosis and should not be confused with atheroma

ATHEROSCLEROSIS OR ATHEROMA involves the intima and may be present in younger individuals Intimal deposits of lipoid material first appear and subsequently undergo softening with a discharge of their contents into the lumen of the vessel An atheromatous ulcer then forms and this lesion paves the way for thrombus formation and embolism The aorta is the most frequent site of atheromatous lesions (see Fig 111)

but their occurrence in the coronary arteries is of considerable more importance to the clinician (see Fig 107) Other types of heart disease (rheumatic syphilitic or congenital) are practically unknown after 70 years of age

While heart disease in old age is very closely allied to arterial degenerative changes by no means all old people show advanced atheroma On the other hand atheromatous changes of extensive degree are sometimes found in young individuals Further discussion of the causes of this early degeneration of the vital arterial structures would lead us into deep water Perhaps it is in some measure due to the speed of living of the person whose arterial inheritance is poor It may appear earlier when tobacco and alcohol are used in excessive amounts Degenerative changes on the other hand may be greatly delayed in those who use these substances in moderation eat sparingly and avoid the hurry and worry of modern existence The speed of living is not so much in evidence among inhabitants of tropical and subtropical countries and this may in part explain the lower incidence of hypertension and arterial disease in these regions

## EXAMINATION

The examination of the heart in older patients is not easy In the first place there is usually a rigid thorax with more than a little accompanying emphysema which makes it difficult or impossible to locate the apex beat For the same reason if percussion is attempted the markings of the cardiac borders that are obtained are usually found to be unreliable An orthodiagram or roentgenogram is quite often needed to determine accurately the cardiac size In the aged, the heart sounds are many times recorded as distant and weak when frequently this change is only a result of the emphysema and in some instances the thickness of underlying tissues of the chest wall Systolic murmurs that have little significance are often present

If we study the anastomotic connections of the coronary tree we note that they increase in number with advancing years This accounts for the smooth course often observed when occlusions develop in old people Frequently coronary changes progress to a marked degree without producing symptoms in aged individuals Even thrombosis appearing as a complication during the course of this disease may be almost harmless Mild degrees of angina in the aged are as a rule easily controlled mechanisms and many of the cases are never seen by the physician for the reason that is usually stated At my age I shouldn t run to the doctor with every ache and pain I expect to have a few

**Other subjective symptoms** that may accompany the aging process are paroxysmal dyspnea acute pulmonary edema and congestive failure Extrasystoles are present in many cases in some instances they may be as common as the remaining gray hairs They are seldom noticed by the

patient and are usually unimportant Often they can be controlled if proper attention is given to the diet and elimination

**Heart Failure** Hypertension produces a left ventricular enlargement and the signs of failure are apt to develop swiftly in some cases following very severe unaccustomed exertion The right sided failure of pulmonary heart disease may add cyanosis and venous engorgement to the picture

**A sclerosis of the heart valves** takes place in old people and at times it may advance to a considerable degree in the aortic valve with calcium deposition particularly if the valve has been previously damaged by rheumatic invasion The structure protrudes into the blood current in these instances, and produces a systolic murmur in the aortic area A more advanced lesion may be accompanied by the typical signs of aortic stenosis (page 127)

**Aneurysms** *While aneurysms are as a whole uncommon in old age, the* dissecting type may occasionally occur and cause sudden death The dissecting aneurysm begins with a split in the intima the blood column usually forcing its way through a necrotic area in the media less often near the margin of an atheromatous patch In this manner the aorta may be dissected in either direction by the column of blood While rupture and death are the usual sequelae in very rare cases organization may take place (page 322)

### Electrocardiogram

Changes in the electrocardiogram may accompany the aging process The voltage becomes reduced the T waves lowered and the QRS complexes as well as the P R intervals show slightly increased duration Changes of this degree are not unusual in senescence Levitt studied 100 men and women in the late decades of life whose histories and physical examinations revealed no evidence of heart disease and discovered that 26 per cent showed definite electrocardiographic abnormalities Willius[408,409,414] reviewed the electrocardiograms of 700 patients over the age of 74 and found that 55 per cent had abnormal tracings

## TREATMENT

The management of the aged patient who has cardiac disease requires *tact diplomacy and skill It is most important first* of all that these patients should be encouraged to keep active and should not be permitted to remain in bed for long periods unless congestive failure or an acute coronary occlusion occurs The physician fails to realize that old people like attention and no matter how gloomy their prognosis they should not be neglected in the daily rounds It should be a constant rule of practice not to disturb their habits of living We should not go to see them in haste with the attitude of a dictator and should not prohibit the old man (or woman) from smoking unless the circumstances in the case indicate the need for the step The same applies to the diet It should be

regulated with tact but much latitude should be allowed unless diabetes is present In this event too severe a restriction of carbohydrate at the start will be likely to be followed by untoward symptoms Fluid should not be restricted unless congestive failure is present If obesity exists we should not hound the patient to start a diet that may send him to his grave but should be tolerant and congratulate him on the good cardio vascular apparatus that he possesses in spite of the handicap of age and weight With this attitude the essential changes in the daily program may be more readily instituted

**Daily Regime** Regulation of the daily regime is all the treatment that is required when mild degrees of structural change in the cardio vascular apparatus are discovered on routine examination Mild dyspnea that is indicative of a decreased myocardial reserve may usually be con trolled by adjusting the exercise allowance (Chapter 20) While the functional capacity of the heart is often reduced in the aged this may not be evident because of the restrictions placed on activity either by advanc ing age or some accompanying degenerative process like arthritis Al though a reasonable amount of exercise is quite beneficial and is responsi ble many times for the ability of old people to carry on unaccustomed or prolonged activity is unwise The ranks of the Blue and the Gray always thin rapidly after the forced marches of Memorial Day or the unaccus tomed activities of anniversary encampments

**Drugs** Great care should be exercised in the drugs that are given to aged patients and too great therapeutic activity should be avoided at all times

Digitalis This is particularly true of digitalis for toxic symptoms are apt to develop very early in the aged While nausea and vomiting may occur following small doses cerebral symptoms are very apt to appear (page 79) Even in the presence of congestive failure digitalis should be given slowly and carefully for older patients require much smaller doses Digitalis should never be given to old people in the absence of congestive failure or fibrillation

Before using mercurial diuretics kidney function should be determined In old men an active diuresis may cause retention so the output should be carefully watched following the administration of any of the organic mercurials If prompt response to the diuretic is not obtained always examine the patient for retention of urine and use an in dwelling catheter when necessary

Glucose is of great value in the treatment of circulatory complications in the aged particularly postoperatively and in the presence of congestive failure It is also useful in combating attacks of paroxysmal cardiac dyspnea or cardiac asthma Given intravenously in amounts up to 400 cc of a 5 to 10 per cent solution it furnishes food for the heart and helps to replace its glycogen store

Morphine is a valuable drug in the treatment of the diseases that are commonly met in the declining years of life We could not do without it

in the presence of inoperable cancer where the pain is great and life expectancy short In heart disease morphine is justified in attacks of dyspnea of the paroxysmal type, but it should be used cautiously Prescriptions for the common and less serious ailments of the aged should never contain morphine, particularly those that are written for bronchitis The drug should always be used with care in pneumonias because of its tendency to produce or increase abdominal distention and to decrease pulmonary ventilation In coronary occlusion morphine should be used in the presence of pain Fenn[99] emphasizes the fact that morphine is more apt to sensitize the vagus and cause reflex coronary constriction and recommends papaverine and atropine In some cases this combination may be entirely effective In other instances it has to be supplemented by the use of small doses of morphine Oxygen administration is of great value in the treatment of coronary occlusion (page 98)

SEDATIVES are many times indicated in geriatric practice, but the dose and the preparation should be chosen with care The acquaintance of the physician with the patient and his habits of living should assist in answering the question as to the advisability of giving whiskey or brandy I have found it valuable in treating cardiac patients of advanced years particularly when the anginal syndrome is present Many times it proves to be efficient when given at bedtime in place of the more commonly used preparations containing barbital

Sedatives of the barbital group have been known to produce mild delirium in many instances, and care should be taken in the amounts that are prescribed for older patients Sodium bromide may be substituted, but is less certain in its action There is no objection to chloral hydrate or paraldehyde and either of these may be used to replace the barbital compounds

**Blood Pressure** Attempts to lower the blood pressure by medication of any type may result disastrously and should not be attempted in the aged since the adjustments to sudden variations in the blood pressure are not as fine in the presence of sclerotic changes (see Case 46) Vertigo and syncope may result or arterial thrombosis in cerebral visceral or peripheral vessels may follow therapeutic efforts of the type that are attended by some slowing of the blood current The increased viscosity of the blood in elderly individuals allows it to adhere readily to the vessel wall and this ever present factor favoring thrombosis should not be given a helping hand by the physician

**Angina** may be completely controlled by regulation of the daily regime If attacks are still experienced small doses of nitroglycerine may be given (page 247) Theophylline ethylene diamine may also be tried (page 270)

**Heart Block** When heart block is accompanied by Adams Stokes seizures, satisfactory management is difficult and the prognosis is usually poor (page 401) Fibrillation in old people is apt to be accompanied by a slow ventricular rate, owing to the decreased power of conduction in the bundle of His The presence of this natural block should make us hesitate to use digitalis if congestive manifestations are absent Quinidine should

never be used to restore normal sinus rhythm in the presence of chronic auricular fibrillation in old people On the other hand quinidine may be life saving in the presence of paroxysmal auricular or ventricular tachycardia

**Delayed Shock** Following even slight accidents old people may develop a syndrome that has been frequently referred to as delayed shock[3, 6] It begins a few days after the accident with symptoms suggestive of a gradual circulatory failure and may terminate in bronchopneumonia Consequently the prognosis of accidents even of trivial character should always be withheld for a few days until this danger period has passed This delayed reaction is not difficult to explain The bed rest restricts breathing and this effect is usually augmented by one or more injections of morphine given for pain following the trauma Stasis develops in the lower lobes of the lungs, and this places extra work on the heart Cardiac failure may be precipitated by infection reaching these lung areas as it often does without much delay unless unusual care is taken

**Surgery** There is an unfortunate tendency on the part of the profession to overrate the danger of surgery in old age When the advisability of operation arises the nature of the contemplated procedure and the cardiovascular status of the patient are usually the first considerations In the common emergencies of old people (acute obstruction acute prostatic retention strangulated hernia) life saving surgery must be recommended However when other operative procedures are contemplated the deciding factor is not the actual age of the patient but the physiologic and pathologic condition of the body as a whole and the circulation and mental state in particular Elderly people should not be denied relief of chronic inconveniences just because of their age for this does not mean that they are poor risks Montgomery and Walters have recently emphasized this point in reporting a series of seven major operations in persons over 70 years of age The patients in this group stood the major procedures very well and all were benefited

If the altered physiologic processes of the aged patient are respected a satisfactory course may be predicted following major surgery It is important to carry out the operative procedures with speed and gentleness and at the same time preserve body temperature and guard against hemorrhage Old people cannot stand blood loss or shock as well as younger patients While shock may appear more slowly reaction to the usual measures for its relief is also a slow and uncertain process However old people usually make good patients for surgery When confronted with an emergency operation they display a much more placid outlook very seldom fear death and show little emotion Moving the aged patient from the hospital to the customary surroundings of the home at the earliest possible time will often be a great aid in the prevention of postoperative circulatory complications

All therapeutic measures whether surgical or medical should always be planned with the age of the patient in mind A multiplicity of remedies for

example even though correctly applied during the course of chronic heart failure may result disastrously where more conservative therapy slowly and cautiously given may be quite efficacious In this respect, when planning the management of a patient of advanced years we should always recall the words of Oliver Wendell Holmes

'so—don t in mercy try
To pump your patient absolutely dry '

# 23

# SOCIAL SERVICE IN THE TREATMENT OF HEART DISEASE

*By* HELEN E HEIKES*

*and*

OLGA TATTERSFIELD§

*The problems involved in the treatment of persons suffering from cardiovascular* disease are common to both urban and rural communities The first problem is the person who is unable to pay the physician s fees and laboratory costs at private rates and it is this type of patient who applies for medical treatment in the heart clinics of the various hospitals of the larger cities Most of these clinics are equipped to render the needed service at a charge within the means of the patient or entirely without cost to him if necessary The clinic physician and his assistants examine the patient make the diagnosis and prescribe the treatment However successful medical treatment depends in some cases on the social factors involved Consequently trained social workers are a necessary addition to the staff in all large hospitals It is their function to help the patient utilize the prescribed medical treatment to the greatest advantage

In a busy heart clinic it is impossible for the physician to be familiar with the personality and social status of each patient The physician outlines the regime of treatment but often obstacles prevent proper execution of these plans Success in treatment may depend on bed rest at home convalescent care a quiet environment and freedom from anxiety better nourishment warmth employment which will not tax the heart and a host of other adjustments The medical social workers concentrate most of their attention on these matters

The first task of the worker is to individualize the patient for the physician Then she supplements the physician s instructions by a detailed interpretation of every item of his diagnosis and treatment It is vitally important for the patient to understand fully his physical condition so that his problems may not be complicated by unnecessary fear of chronic invalidism or sudden death The social worker can also assist the patient in adjusting personality problems frequently the patient has to be helped to express his fears resentments and bewilderments before treatment can be carried on successfully The patient can also be aided in utilizing his community resources with regard to school his occupational and recre

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ational life and the development of all of his capacities as much as possible within his limitations

In looking further into the many aspects of the social worker's role in combating heart disease let us see what can be done about the child who has rheumatic heart disease Although the medical needs are met by clinic or family physician what other factors are involved in his care? Damp, over crowded homes poor physical hygiene, and lack of proper nourishment are certainly not conducive to combating rheumatic infection The child's school life is especially important Children with heart disease should have the maximum opportunity for education with due attention to vocational adaptation in order that they may receive training for gainful occupations that will not prove too great a strain upon the heart muscle Where school adjustments are necessary, the co-operation of the medical organization of the public school system as well as of the principal and teachers must be secured These persons should be acquainted with the physical condition of the child and with the necessary adjustments that will enable him to carry out his school work successfully Sometimes it is advisable to request the use of the school elevator or to transfer the child to a classroom on the lower floor Perhaps the child can easily climb the necessary stairs if allowed to take his time going up and down either before or after the other children Sometimes permission must be obtained to use recess periods for rest periods instead of for the purpose of recreation

At home the child may be the victim of either over anxiety or indifference on the part of the parents Other factors that hinder progress are lack of parental control family friction and the child's resentment of and refusal to accept exercise restrictions also the use by the child of his physical disability as an attention getter in his home and social group Often the social worker must establish a direct relationship with the child in order to assist him in working out his problems Where the home environment is such that the child cannot have adequate care placement in a convalescent hospital or foster home may be necessary

> The physician or clinic is but a hub in the wheel of cardiac treatment and a great part of the problem of the cardiac child is the problem of the home and social and school life'[31]

With the adult we have the problem not of prevention but of adjustments necessary to keep the patient in such condition that he may lead a useful contented life within his limitations Here again the fear element must be overcome not only by the family but by the patient himself Fear of lack of ability to provide for himself and for his dependents fear of chronic invalidism or of sudden death often retard progress The readjustment of the economic status of the patient is also an important social factor in recovery Very often the mother of a family needs help in her daily work convalescent care must be provided and in cases where the patient cannot return to his regular work sheltered employment and occu

pational therapy can be used to aid the process of rehabilitation In some instances the patient can return to his former occupation provided some minor adjustments are made

In the life of the cardiac patient recreational opportunities are important for both adults and children It is vital in making the adjustment to the disease that the patient meet his associates on the basis of what he can do instead of on the level of what he is not permitted to do along recreational lines Too often the cardiac patient tends to become withdrawn because he is too conscious of his physical limitations and it requires skillful teamwork by the physician and the social worker to help the patient to sub ti tute a more limited but satisfying type of recreation for the more active forms he once enjoyed

The following case histories illustrate some of the situations in which the social worker functions in helping the patient to solve the problems that hinder medical treatment *

## ILLUSTRATIVE CASES

**Case 110** Henry K age e ght was referred to a heart clin c by the school doctor A phys cal examination revealed a funct onal heart murmur and the patient was placed n Class F After the examination his moth r asked the do tor the result. He told her b iefly that there was no cause for worry but that he would l ke to re-examine Henry in six to e ght month Four months lat r Henry contracted pneumonia and was admitted to the hosp tal When the social worker interv ewed th mother relative to the data the doctor needed to complete the pi ture of his patient she learned that Henry was s llen and d sobedient and had been absent w thout leave from school several times The mother said that she had restri ted his exercise because he had heart trouble forbade him to run roller skate swim play ball etc The doctor had told her not to wo ry but she had heard of people who dropped d ad suddenly of heart disease and the doctor would not want to exam ne Henry again if his heart was all right The school do tor also had sa d that Henry had heart t ouble

The social worker was able to expla n to this bew lder d mother that Henry had no o gan c heart lesion that there was no reason to restrict the boy s exercise She also made clear why the doctor had r quested the re-exam nat on The mothe accepted the explana t on and allowed H nry to assume h usual ct vities after he had recove ed from his ill ness When he returned to cardiac clin the orker w nt with him and his mother to the docto and at the examination Henry was discharged from the clin c The mother had an opportunity to talk with the doctor and later w th the social wo ke and said th t she understood and was entirely satisfi d Needless to say Henry s behav or problems d d not return and he was leading a normal l fe when last seen

Discussion This illustrates the average person s conception of heart di ease and the damage which can be done to the individual unless he has adequate interpretation and help from the physician and the social worker

**Case 111** Ed th Y age 11 Diagnos s chorea Class F

Ed th w s bro ght to the hosp tal acutely ill ith chorea nd was adm tted The mother distu bed the hospital administration by walking up and down in f ont of the

* We h to thank the S cial Servi e D pa t ents of the Graduate P nn ylvan a St Ch t ph r nd Woman s College Ho pital of Philadelph a and the Placement Serv ce of the Philadelph a Health Council a d th T berculosis Comm ttee for the case r cord used in this chapte

hospital several hours each day although she could visit only once weekly On being questioned, the mother stated that she could not help herself The doctor explained to the parents the child s diagnosis and told them that she needed prolonged rest that there was no heart damage then and that complete recovery was likely The case was referred to the Social Service Department by the physician, and an appointment was made to see the mother Both parents were very emotional The mother had hyperthyroidism and the husband was a stubborn German type The father blamed the mother for the child s illnes and th mother blam d th lack of proper food The worker had to make the parents understand that the hospital did not consider that Edith was ill because of her care at home that the cause of chorea is unknown, but that it can b controlled by proper treatment The parents wished to take the child home against advice several times but after an interview with the social worker they revised the r decision

In view of the friction in the home the doctor recommended a long period of convalescent care for the child After much work on the part of the social service department and the physician the child was allowed to go the parents at first refusing because they did not wish Edith to be away from home any longer One month later Mr V informed the social worker that she was taking Edith from the Children s Heart Hospital since her husband was out of work and could not pay the board rate The social worker interviewed the hospital and arranged that the charge for board be cancelled so that Edith could remain as long as necessary Upon Edith s return to h art clinic after discharg from the convalescent home, her condition was good and the parents followed the doctor s recommendations for her home care as interpreted by the social worker

Discussion In this case the value of the social worker is seen

(1) In acquainting the parents with the nature of the child s illness and so preventing withdrawal of the patient from the hospital before convalescence was complete

(2) In arranging for continued convalescent care at the Heart Hospital

(3) In interpreting to the mother the do to s recommendations for the final home care which are at present being carried out

**Case 112** Roger M age 11 years had be n a pati nt in heart clinic for several years suffering from rheumatic heart di ease His exercise was quite restric ed and he had little opportunity for school because of long periods in the hospital and equally long periods of bed rest in convalescent hospitals This enforced inactivity bred resentment and he became a thorn in the side of the authorities at several convalescent homes When a third hospitalization became necessary Roger broke out into violent rebellion and temper tantrums

The medical social worker and the physician reviewed the situation and wha it meant to Roger The boy s home life was troubled and insecure The father a weak and inadequate person drank to excess and sough to maintain his status as head of the house by bullying his family The mother a wornout household drudge tried to work with the hospital but her efforts were confined to nagging Roger about over st pping his exercise prescriptions Two older sisters added their voi es to the family chorus of d sapproval of Roger s conduct, which merely serve l to increase the boy s resentment against the family the hospital and the world where all forces seemed to be gathered against him

It was evident that Roger could not reach an adjustment in such an environment Likewise a return to a conval scent home held no solution to his problem since h was fighting institutionalization and restriction and was having little opportunity for education Foster home placement seemed worth trying and the situation was discusse l with an excellent local agency They finally agreed to make placement upon a med al basis Roger continuing his medical care in heart clinic as before

This plan was d scu sed with Roger and his parents as a med cal plan recommended by

the physician and they agreed to it Roger was accordingly placed in a foster family gradually resumed activity went to school and was seen regularly in heart clinic Throughout this period away from home he was visited by his family and his relationship to them was kept always before him It required several years and three changes of home before Roger was able to work through his problems and make a real adjustment to his disease but he finally was considered well enough and sufficiently balanced emotionally to return home where he has been for several years He managed to regain his standing in school and now enjoys life with a much increased exercise tolerance In addition he has a recognized place in the family and community

Discussion This case shows how serious behavior problems may result at times from over institutionalization and from lack of understanding in the home Individualizing the boy in a foster home with understanding foster parents focused his attention upon his possibilities instead of his handicap so that he realized himself as a part of society instead of one cut off from his fellows by an arbitrary restriction

**Case 113** Edward A colored age 11 years was referred to the heart clinic from medical clinic where a diagnosis of rheumatic heart disease was made The boy was underweight and malnourished and had developed behavior problems at school The home background was lacking in what we consider good child care Edward was an illegitimate child his mother was employed by day and was often absent from home at night Edward was irregular in his school attendance was disobedient disturbed the classroom by antics designed to get attention pilfered small amounts of money from his mother and ran the street until late at night The physician in heart clinic referred the problem to the social worker and the boy to neuropsychiatric clinic

Edward was found to have an I Q of 114 and although a year younger than the other children in his class at school was able to do the required work He had lived with his maternal grandmother until her death and had been with his mother only ten months and was practically the only colored child in the neighborhood His mother gained a degree of understanding of the factors unbalancing the boy s life and decided to give up her work and stay at home a paramour having come into the picture who was willing to support mother and child and for whom Edward had developed a real affection as a father substitute Edward was transferred to another school and did well for a time because teachers and principal interested themselves in his adjustment When they relaxed their attentions Edward s behavior problems gradually reappeared His mother came to the social worker for help because Edward was fighting with the other children missing school stealing and she thought his health must be poor and that there was something wrong with his mind The situation was discussed with the physician on the basis of the child s heart condition while the need of better home supervision and a plan for a foster home placement was worked out with the mother The services of the placement agency were enlisted and Edward was established in a foster home where the foster parents had a good understanding of boys their needs and problems Since placement Edward has gained considerable weight his heart has definitely improved and behavior problems are no longer in the picture He gets along well with other children and the school authorities have no complaints He maintains contact with his mother who visits him frequently

Discussion This picture shows a bright alert colored boy suddenly thrust into an unstable home atmosphere after the death of his grand mother who had reared him Lack of parental supervision unwholesome family relationships and isolation in a community where white people pre dominated gave rise to the personality difficulties which manifested them selves in antisocial behavior Efforts to help Edward to become adapted to

this environment failing foster home care was carried out and the boy responded to love and a stable home environment, although maintaining contact with his mother

**Case 114** Julia G age 1 Diagnosis rheumatic heart disease with mitral stenosis The findings of the cardiologist and the recommendations for convalescent care were discussed by the social workers with the parents and a plan was formulated wher by Julia was admitted to the Childrens Heart Hospital She remained there for four months returning home at the end of that time

Julia lived with her family of seven persons in a congested section of the city in a small house which was in fairly good condition Her parents were Irish American took an intelligent interest in Julias care and tried in every way to work with the doctor and social worker After Julias discharge from the Heart Hospital she came regularly to clinic and the parents tried to limit her activity but she was restless and showed a continuous temperature and weight loss that finally necessitated a second hospital admission Convalescent care was again advised the parents agreed and Julia was re admitted to the Heart Hospital where she stayed for eight months She returned to school and was placed in a nutrition class at the request of the doctor in heart clinic but again she lost weight and did not do well in classes Her father was so fearful of school by this time that conference with the physician of the Board of Education was arranged for him by the social worker and the child was allowed to remain at home for the rest *of the school year (three months) In July she was sent to the Childrens Seashore House* f r three weeks

Julia started school again in September did well and reported regularly to heart clinic during the next year and had few set backs She soon began going to dances and lived a normal social life Her cardiac status improved during the next few years At the age of 19 she married Her first baby was stillborn When her second baby was expected she came to the social worker with the request for assistance in obtaining prenatal care and hospital delivery Satisfactory arrangements were made for her hospital delivery Following this she had two other children has no symptoms referable to the cardiovascular system and now is leading a happy normal life

**Discussion** This case is illustrative of the value of intelligent participation by parents and child in a regime of medical care that is formulated by the physician and carried out with the help of the social worker Long term convalescent care and supervision helped to arrest rheumatic activity in this case and the patient led a normal happy life within limitations that were understood and accepted

**Case 115** Eleanor M age nine was admitted to the heart clinic because of malnourishment and active rheumatic heart disease Investigation of the home environment showed that her mother and father were constantly bickering and had no conception of the childs needs placing upon her all the responsibility for rest and care Since manifestly the child could not progress under such conditions convalescent care at the Childrens Heart Hospital was arranged In a few months the parents demanded the childs return as they were on relief and sought this means of *having their grant increased* The parents demands were discussed with the chief of the cardiac clinic and he was willing to have Eleanor return home provided she would be kept in bed Eleanor accordingly returned home and a visiting nurse was called in to give her bedside care Milk and additional food for the child were provid d by a private family agency The nurse soon reported that she found that the continuous quarreling of the parents was affecting the child and retarding her recovery The social worker arranged an interview for the father with the chief of the cardiac clinic who explained to the father the importance of quiet and restfulness for the child at home wh le the social worker brought out similar points in a talk with the mother The parents agreed to give

Eleanor a room to herself away from family disturbances and an occupational therapist was also sent in to help her during her enforced rest The child is improving steadily at this time and her exercise tolerance has increased

**Discussion** This case history again illustrates how family friction can retard a child s recovery and how the social worker can help both parents and the child to a better adjustment

**Case 116** John H a boy of 16 was referred to the Social Service Department of the hospital where he was attending heart clinic by the clinic physician to see what could be arranged to enable him to go on with his art work at which he showed great aptitude The boy s family consisted of an indolent father who would not work a mother who did day s work when she could get it and a sister of 17 who worked regularly and practically supported the entire family The father wanted John to leave school and get a job to help in the support of the family The doctor felt that John needed sedentary work as he had very definite heart damage and could not stand any form of manual labor The social worker discussed this with the family and after many visits persuaded the father to let John go on with his studies provided a scholarship could be secured for him She then took this problem to the local heart association and together they secured a scholarship for John in art school An interested person offered to pay for the supplies he needed and his sister consented to pay his carefare and incidental expenses

John spent three years at art school and did very well he received prizes for his prints and honorable mention for his charcoal work After he finished his training a position was secured for him with a firm of engravers but this work proved too arduous and his health began to suffer He was forced to give it up However he proved to be an energetic boy and soon secured some art work on his own time He is at present free lancing and making a good living for himself and his parents The sister has married and left the home and John is supporting himself and his parents adequately

**Case 117** William G age 19 when referred to cardiac clinic by the eye clinic was found to be suffering from advanced rheumatic heart disease The social worker interviewed William and at his request referred him to the Bureau of Rehabilitation as his heart condition prevented laborious work The bureau gave no encouragement to the patient about training for work within his activity limitation He was dependent upon his sister who was resentful of having to provide for him and their mother and made life very unpleasant for both Through the efforts of the social worker in heart clinic a training course at a school of occupational therapy was secured for William and a job provided at the end of the training course by the Shut In Society At this time he developed pneumonia and was admitted to the hospital He was greatly worried about his job until the social worker told him that she had found that it would be open for him when he recovered He returned home and to work Later he was upset and despondent when his mother became ill After her recovery the patient was in such a poor condition through anxiety that the clinic physician recommended convalescent care and William agreed to go to a convalescent home In the meantime the sister who was the disturbing element in the family moved away and William and his mother now live a much happier peaceful life although he is at the present time still unable to go back to work and he and his mother are living on a relief grant The clinic physician feels however that if William continues to improve under the better home condition in time he will be able to resume work and be self supporting

**Case 118** Henry G 29 years old a patient in heart clinic was referred to the Social Service Department by the chief of clinic to see if some plan could be worked out whereby Henry could earn a living for himself and his family which consisted of his wife and three small children His physician stated that the work he was doing in a leather factory was much too strenuous The social worker referred Henry to an employment bureau for the handicapped and the placement worker there finally secured a job for

*the patient in another factory making small suitcases and bags It was a finishing job, and Henry could sit down while working He has been with this firm for about even years and is doing well physically as well as economically*

**Case 119** *William N an unemployed laborer of 66 suff ring from hyp rtensive cardiovascular disease was referred to the Social Service Department by a clinic physician to see if some work could not be secured for him to do at home The patient s heart was not able to stand the strain of employment outside the home but the physician felt that he needed something to keep him occupied The patient lived with a married daughter and her family who had quite a struggle to get along financially The social worker made arrangements for an occupational therapist to visit William and teach him to do some handicraft He was interest d in learning to make hooked rugs and soon became very proficient working on a frame placed in front of his chair at an angle so that there was no strain on his arms William worked at rug making for about three years filling orders that the occupational therapist helped to secure for him and earned between $15 and $30 a month This money was a great help to his daughter as he was able to pay hi board and he was happy and contented in his work William died three years later but we believe that this work probably prolonged his life, since he had been restless and unhappy before he undertook it*

**Case 120** *Mrs T This patient a widow of 4 was a foreigner who spoke very good English She was referred to heart clinic from the general medical clinic It was found that she had an advanced cardiac lesion and could not continue the domestic ervice in which she had been engaged She had no relatives in this country and no one to help her financially Upon th physician s re ommendation she was admitted to the hospital for a period of several weeks and then sent to a convalescent home in the country for a month s stay The social worker meanwhile got in touch with a family agency and succeed d in g tting a t mporary w kly grant Th worker then referred the patient to an employment agency for the handicapped A vocational guidance test at the local university proved that she was capable of business training A scholarsh p to a business college was then provided and a room was secured for her rear the busine s school The patient quickly and successfully completed the course of study and a job was secured for her by an employment agency The family agency withdrew its grant as the patient became self supporting At the la t report the patient had be ome a valued member of an office staff was happy and satisfied in her work and had entered into the social activities of her community*

Discussion Had the services of the social worker not been available, this patient could not have made use of the valuable community resources as she knew nothing of such services Her medical treatment, therefore could not have been satisfactory either to doctor or to patient

The foregoing case histories are illustrative of the many social factors which complicate life for the cardiac patient and handicap medical treat ment They show also how the physician and the social worker function in the interest of the patient The physician in the conduct of a busy clinic must necessarily relegate the social treatment of the patient to the medical social worker From this point there must be close co-operation be tween worker and physician in the patient s interest The physician s re sponsibility is to outline treatment, the social worker duty is to individ ualize the patient to the physician to consult with the phy ician and to keep him informed as to the progress of the medical social plan

The social worker has an allied responsibility to the patient to inter pret the diagnosis and medical treatment to him to help him to use hos pital and community resources to further his medical treatment and to

aid him in working out the personality problems which may be blocking his adjustment to a physical limitation She can function however insofar as the patient is conscious of his need and desires her help Often she sees more than one problem as she analyzes the patient s social situation but she can work only with the problem that the patient sees as the immediate one

This physician social worker relationship is necessary in clinic work As stated before the physician in rural or small urban communities has the advantage of a closer relationship to his patients and a much less complicated community organization He goes into the homes of his patients and sees them as individuals in their respective family and community groups In these instances the physician can more easily help the patients to work out their adjustments as he has ready access to community resources His personal interest in helping his patients to make adjustments and to solve the problems that hinder medical treatment is a part of the art of medical practice It can never be successfully replaced by bureaus or agencies under state control

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# AN INTRODUCTION TO THE STUDY OF ELECTROCARDIOGRAPHY

## HISTORY

It has been stated that the history of any science is the science itself Consequently there is no better means of approach to the subject of electrocardiography than a *review of the steps by which it reached its present* state [96] From small beginnings in the laboratory to the ultimate perfection of an instrument of great value at the bedside the story winds through nearly 150 years The contributions of many investigators made possible the final work of Einthoven who introduced the instrument into clinical medicine

Electrocardiography is based upon the fundamental physiologic fact that the contraction of a muscle is accompanied by a minute electrical current Therefore if we desire to go back to the beginning we should examine the earliest experiments in electrophysiology These were made by Luigi Galvani toward the end of the eighteenth century Of course the indefatigable John Hunter studied animal electricity in 1773 using the torpedo or electric ray fish This peculiar property of the fish was not discovered by him however for we read that the Romans were aware of it and for this reason made some use of the fish therapeutically Caldani likewise performed several experiments on the electrical stimulation of the cerebral cortex as early as 1784 However Galvani's work because of its completeness and conclusiveness marks the real beginning of our knowledge

Galvani was a distinguished professor of anatomy at the University of Bologna and early in his career became very much interested in animal electricity Quite by accident in 1791 he placed a dissected frog on a laboratory table near an electric machine It was a fortunate circumstance but the interpretation of the result had been waiting for a man of Galvani's insight and ability As an assistant lightly touched the nerves of the frog's leg with the point of a knife Galvani observed that the muscles were thrown into vigorous contraction

Galvani was very much interested and curious and he was determined to find out if lightning would produce the same effect One day during a thunder storm he dissected a frog and attached it to a conductor He connected the feet of the frog to a wire which he grounded into the water of a nearby well Galvani observed that

> The results came about as we wished As often as the lightning

broke forth the muscles were thrown into repeated violent contractions so that always as the lightning lighted the sky the muscle contractions and movements preceded the thunder and as it were announced its coming It was best however when the lightning was strong or the clouds from which it broke forth were near the place of the experiment

This experiment opened up a wide field for investigation Galvani later discovered that when one of his frogs was placed on a metallic plate and the hook piercing its spinal column was brought in contact with the plate a twitching occurred He repeated the experiment and in place of the metal plate he used glass and the twitching disappeared He also learned that if the frog rested on a glass plate and he touched both the nerve and the muscle with a bent rod consisting of two different metals prolonged convulsions followed In these simple experiments Galvani produced the first cell in history for generating electricity

The next advance was made by Carlo Matteucci who first demonstrated the rheoscopic frog effect (1842) In this experiment if the sciatic nerve of one leg of a frog is placed upon the muscles of the opposite leg the muscles of both legs may be made to contract by simply stimulating the sciatic nerve on the normal side The essential point for us to remember about this contribution in connection with electrocardiography is the fact that the second muscle was not stimulated directly by the current applied to the nerve but indirectly by the current of action generated in the muscle in consequence of its contraction

The time soon arrived for an application of this knowledge to the heart In 1856 Albert von Kolliker and Muller discovered that the tiny frog s heart exposed and contracting on their laboratory table produced an electric current which accompanied each beat This fact they established but at the time very little practical use could be made of it because of the crude apparatus available for additional investigation

It was not until 1878 that further progress was made by two English physiologists Sanderson and Page These workers successfully recorded for the first time the minute heart current described by von Kolliker and Muller by means of the capillary electrometer This instrument consists of a column of mercury in a vertical glass tube the end of which dips into sulfuric acid These investigators noted that if an electric current no matter how small disturbs the relation between the mercury and the acid the slender column of mercury moves to a new position in the tube This response of the mercury column to the electric current was photographed by Sanderson and Page on a moving sensitive plate

However even the physiologists found that the capillary electrometer was a delicate temperamental instrument It was no easy task to make it operate successfully and at times it must have sorely tried the patience of the laboratory workers Then too the hearts under investigation had

to be connected directly to the instrument which made the study a matter of laboratory interest only

In 1887 Waller made the important discovery that the heart s currents could be demonstrated without opening the chests of the laboratory animals He merely connected the outside of the body to the capillary electrometer by electrode These wire connections were later to become known as 'Leads ' Waller immediately applied this idea to man and found that here too the delicate heart current could be led off the arms and legs of his subjects

So far in our survey we see that the scientists had shown a small current to be present when the heart contracted they had found a way to measure it crudely and Waller showed that the current could be led off the arms and legs of the subject However, the troublesome capillary electrometer remained The acid and the mercury had their faults and shortcomings The mercury possessed inertia and the curves of its movements, when photographed were not true curves The hard pressed physiologists had to correct their experiments for the mercury inertia by mathematical computations Surely the practitioner of medicine could not be expected to carry out such work so this valuable method of study had to remain in the laboratory

*The years were quickly passing and the century was almost spent* Many discoveries of more dramatic and of a more practical nature were taking the attention of the physicians New germs were being described antisepsis was coming into vogue in fact medical discoveries were pouring in on the bewildered doctors from all quarters with the rapidity of corn popping in a pan Consequently the progress made in the registration of the heart s current remained far below the clinical horizon

With the turn of the century the man destined to bring the method out of the shadow arrived on the scene The year was 1903 the place Leyden Holland the man Willem Einthoven whose final contribution made electrocardiography a clinical reality His work enabled scientists to discard the capillary electrometer and use in its place for recording the heart s current the much more rugged and reliable string galvanometer This instrument had already been invented by J S C Schweigger of the University of Halle but Einthoven perfected it and applied it to the measuring of the electric current generated by the heart s contraction

He found that the delicate quartz string contained in the galvanometer moved more quickly and did not have the lag of the mercury This observation gave us at last a practical method of electrocardiography With Einthoven s use of the galvanometer in place of the capillary electrometer the journey of the method from the laboratory to the bedside took just a few years The scientific study of diseases of the heart then began in earnest

The original apparatus of Einthoven was very large and cumbersome The magnet was of considerable size and the string that was mounted be

tween its poles was an intricate mechanism The original instruments took up considerable space and the details of their operation discouraged the ordinary clinical worker For this reason few hospitals had electrocardiographs prior to 1905 06 About this time Sir Thomas Lewis began to use the electrocardiograph in England and the popularity of the method at the end of the first decade of the present century was largely a result of his efforts As the century grew older the instrument was simplified and more and more was learned of its usefulness The improved instrument was made to occupy less space in the laboratory until more compact and easily portable electrocardiographs for bedside work became a reality

Today the clinician leans heavily on the electrocardiograph In some cardiac conditions a tracing is essential before a final diagnosis can be made Moreover the electrocardiogram tells a great deal concerning the progress of patients and guides the administration of certain drugs Its future is bright The tiny current produced by the frog s heart in van Kolliker s laboratory in Wurzburg in 1856 opened up a large field for scientific study and research which is broadening year by year The ultimate extent of its progress is a matter of pure conjecture as new depths are being plumbed and new uses are being discovered for the method by clinicians as well as by laboratory workers

## THE APPARATUS

Let us now examine the essential features of the modern electrocardiograph (Fig 164) The lamp (L) sends a beam of light through a condens-

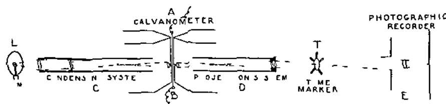



Fig 164 A simplified diagram showing the essential parts of an electrocardiograph of the string galvanometer type See text for description

ing system (C) This is focused on the galvanometer string (A B) stretched in an electromagnetic field between the poles of two powerful electromagnets When the heart current of the patient is passed through this fine quartz string the string moves across the gap at right angles to the lines of magnetic force and its shadow is magnified as it passes through the projection system (D) and falls on the moving strip of film in the camera at E At T a time marker interrupts the beam of light at intervals of 0 04 second and these vertical markings appear on the developed strip of film (Fig 165) This is the principle of the instruments that contain a string galvanometer For a more detailed description of the construction of the

various types of electrocardiographs the reader is referred to the special texts on the subject [6, 7]

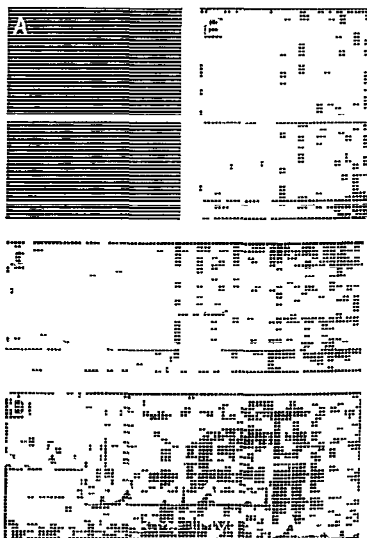



FIG 165 A Shadow of the string The time marker is not running B Same with the time marker running Note the presence of vertical lines that indicate the time markings C Standardizing string tension before introducing the patient into the circuit. Note the deflections of the string caused by one and two millivolts D Normal electrocardiogram (Lead I) Note the standardization The introduction of 1 mv deflects the string exactly one cm

In the early days a very large room in the hospital was assigned to the Heart Department to accommodate the equipment Electrocardiographs were big and cumbersome repairs were difficult and a finished record was

an achievement Today most models are rugged easily operated by a technician and seldom present the mechanical difficulties that made the early workers in this field true pioneers Figure 166 shows a model in common use today A glance will show the general principle of its construction since its various parts are designated by the same letters used

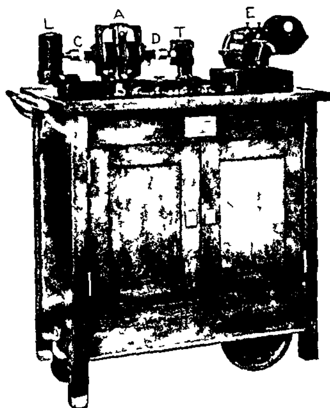



FIG 166 Modern mobile type electrocardiograph containing string galvanometer The letters correspond to those of Fig 164 (Courtesy Cambridge Instrument Co)

in the schematic drawing Recently smaller more compact models that can be readily carried to the bedside of the patient have been perfected (Fig 167)

There is a difference in the construction of some of these instruments that deserves mention Perfection of the amplifier tube has made it possible to magnify many times the current derived from the patient This permits the use of a less delicate galvanometer Instead of photographing the motions of the string these newer instruments record the motion of a beam of light reflected from a mirror contained in the

galvanometer (Fig 168) At present there are several models on the market that employ this principle They are small, serve readily at the bed side and appear to be as suitable as the original models containing the more delicate string galvanometer The electrocardiograms obtained from the two types of instruments are identical, the amplification of the heart current *apparently does not* distort the individual waves A recent addition to the electrocardiograph that has appeared is a compact system for recording the heart sounds simultaneously with the electrocardiogram (pages 23 584)



Fig 167 A bedside tracing using portable model electrocardiograph (Courtesy Children's Heart Hospital and Cambridge Instrument Co )

*An addition to one of the amplifying types of electrocardiographs* recently placed on the market is the 'Cardioscope' This attachment makes possible the reproduction of the image of the electrocardiogram on a moving fluorescent screen or drum (Fig 169) The electrocardiogram can be seen by looking through the window of the instrument and the reading may be made at once Time and amplitude lines are thrown on the screen when desired by means of a switch The Cardioscope makes it possible to secure a constant viewing of the heart s action during the course of a long surgical operation or animal experiment and consequently saves considerable film A tracing however may be recorded at any time if the physician detects an interesting event in the electrocardiogram on the moving drum In cases of suspected coronary occlusion this instrument also

Fig 168 Diagram illustrating principles of electrocardiograph of the vacuum tube type The current from the patient passes through amplifier (1) and then to a galvanometer of special design ( ) The galvanometer contains a mirror which reflects a beam of light from source (3) As the galvanometer responds to the amplified heart current it causes the beam of light to move across the film in the camera (5) (Courtesy General Electric Co )

Fig 169 The cardioscope (Courtesy Sanborn Company )

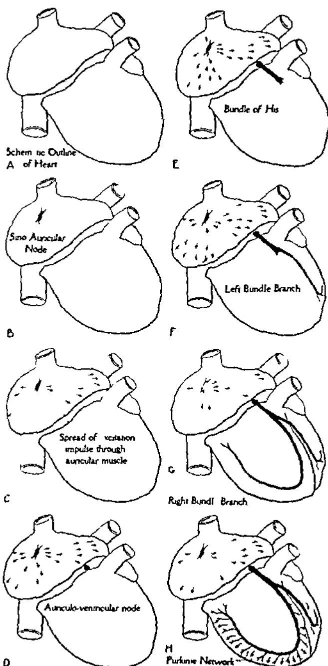



FIG 170 Schematic diagrams representing component parts of the con luct on system of the heart See text for explanation

enables the investigator to note the electrocardiogram obtained by using various points of contact on the chest wall and to record on the film only the complexes that appear to be most useful in the diagnosis

## PHYSIOLOGIC PRINCIPLES

The chambers of the heart contract in sequence following an excitation wave that passes down the specialized tissue of the conduction system (Fig 170) The impulse for cardiac contraction originates in the sino

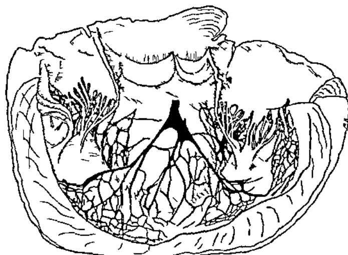


FIG 171 India ink injection of beef heart showing the conduction system

auricular node (synonyms sinus node the Keith Flack node the S A node the pacemaker) This is a vascularized island of special tissue situated high up on the posterior wall of the right auricle (B) The impulse passed from here into the muscle of the auricle There is no special path way for conducting this impulse through the auricular musculature consequently the wave spread equally in all directions (C)

Another clump of specialized tissue the auriculo ventricular node (synonyms A V node node of Aschoff and Tawara), next receives the impulse (D) From here it travels down the bundle of His (E) and is distributed equally to each ventricle through the right and left branches of the bundle (F and G) finally arriving at the terminal *ramifications of* the system the Purkinje network (H) lining the inner ventricular wall The spread of the impulse is now complete

Although the human heart does not lend itself readily to a demonstration of this conduction system the ox heart with the proper technic and a little patience may be injected to show the entire system If the fresh

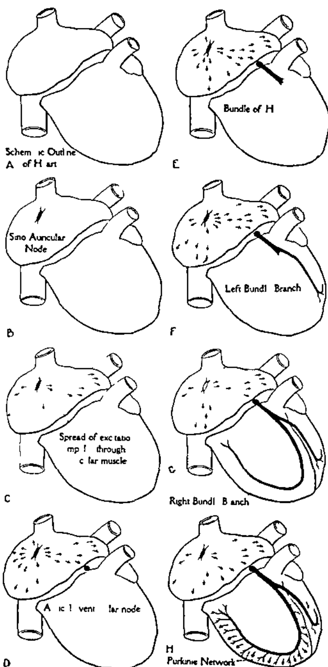



FIG 170 Schematic diagrams representing component parts of the con luction system of the heart See text for explanation

enables the investigator to note the electrocardiogram obtained by using various points of contact on the chest wall and to record on the film only the complexes that appear to be most useful in the diagnosis

## PHYSIOLOGIC PRINCIPLES

The chambers of the heart contract in sequence following an excitation wave that passes down the specialized tissue of the conduction system (Fig 170) The impulse for cardiac contraction originates in the sino-

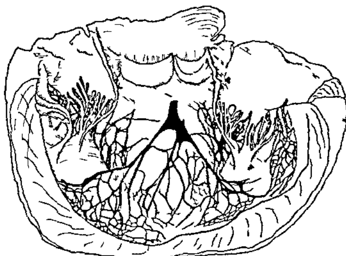


Fig 171 India ink injection of beef heart showing the conduction system

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Although the human heart does not lend itself readily to a demonstration of this conduction system the ox heart with the proper technic and a little patience may be injected to show the entire system If the fresh

ox heart is allowed to stand for 12 hours at room temperature the slight shrinkage that follows early degeneration about the specialized conduction tissue will permit the passage of injection fluid The needle of a syringe containing 5 cc of India ink is then inserted into the upper part of the

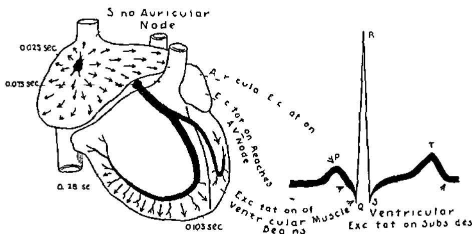



FIG 172 Diagram illustrating relationship between the spread of the excitation impulse and the electrocardiogram The figures represent the time intervals

bundle A slight amount of pressure forces the ink along this dead space, and the whole conduction system will be strikingly outlined (Fig 171)

The waves of the electrocardiogram are produced by the spread of the excitation impulse and are in no way related to the strength of the ensuing cardiac contraction These waves were originally named by Einthoven

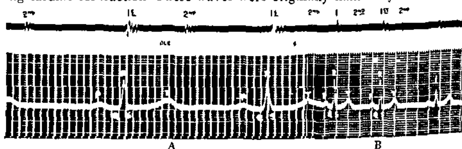
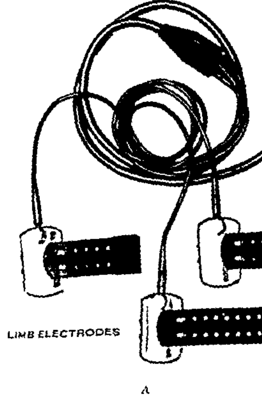



FIG 173 Sound tracing and electrocardiogram simultaneously recorded by Sanborn Stetho cardiette A Speed of paper 75 mm per second B Speed of paper 25 mm per second (Courtesy Sanborn Company)

in purely empirical fashion P Q R S and T Figure 172 shows the relationship between the spread of the impulse and the electrocardiogram As the electrical excitation passes through the auricular muscle the P or auricular wave is inscribed in presystole The string then returns to the base line With the upstroke of the R wave the auricular cycle (or auricular complex) ends The ventricular cycle (or complex) usu



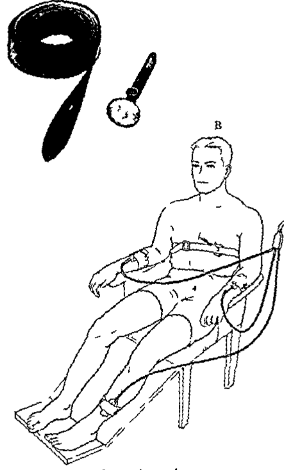




Fig 174 A Electrodes for direct and indirect leads B Electrodes in place

ally opens with a small initial downward dip of the string or Q wave, followed by an upward R wave Again the string returns to the base line At this point a small dip below this level may be observed This downward deflection is the S wave A broad blunt upright wave is next inscribed, this is known as the T wave Rarely, an additional smaller, and usually unimportant wave may follow the T wave This is known as the U wave and is produced by the events of early diastole (see Fig 175)

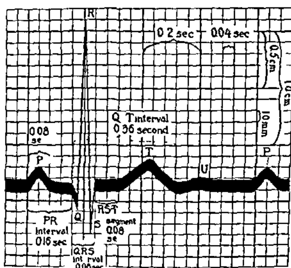



Fig 175 The electrocardiogram single lead (From Essentials of Electrocardiography Ashman and Hull Macmillan Company N Y)

In Fig 173 we will see the relationship between the electrocardiogram and the heart sounds of a normal subject Note that the peak of the R wave is recorded before the first sound of the heart is heard at the apex The second sound of the heart immediately follows the T wave

As previously stated any two parts of the body with the heart between them may be chosen as points of contact for leading the current of the heart from the body These leads are three in number and for convenience the arms and the left leg are used The waves of the electrocardiogram vary in relation to the location of the electrodes on the body surface Lead 1 is obtained by placing the German silver electrodes on the arms (Fig 174) This is usually done by rubbing the skin at the point of contact with a small amount of a special jelly Lead 2 represents the curve obtained when the current passes through the string galvanometer from the right arm to the left leg and lead 3 results when the left arm and the left leg are connected Using three leads in each case gives a better tracing of cardiac events than any single lead Additional precordial leads are obtained by *placing the electrodes directly on the chest* These special direct leads will be considered in detail later (page 637)

## THE ELECTROCARDIOGRAM

We will now turn our attention to a study of the details of the electrocardiogram First of all it should be noted (Fig 175) that the smallest squares in the blocks represent 0 04 second in the horizontal direction In the vertical direction each block represents a potential difference of 0 1 millivolt Note that the string moves over ten of these small squares when the operator throws one millivolt into the circuit (see Fig 165) In other words each electrocardiogram is standardized so that ten small blocks in the vertical direction (or 1 cm ) equal a potential difference of one millivolt Consequently in all electrocardiograms no matter where they are taken the voltage of the waves can be determined accurately The voltage or amplitude of all waves is measured from the top of the base line to the top of the deflection If we are dealing with a downward deflection we measure from the bottom of the base line to the lowest point of the deflection The duration of the waves and intervals can be accurately measured on the horizontal base line

## TECHNICAL FAULTS

For detailed technical data concerning the recording developing and filing of the electrocardiograms the reader is referred to one of the special

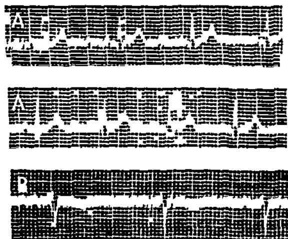


Fig 176 Artefacts in the electrocardiogram caused by A Nervous patient (not relaxed) Middle strip (A) and last strip (B) show improper elimination of electrical interference in vicinity of patient

treatises on the subject However the physician should become acquainted with a few details of the technic that have a direct bearing on the form of the record he receives A laboratory report of any kind to be of clinical

value must be the work of a careful and skilled technician The electro cardiogram is no exception to this rule

The room in which the tracings are taken should be quiet and apart from the main thoroughfare of hospital or office It should be kept com fortably warm in the winter and should have no telephone connections

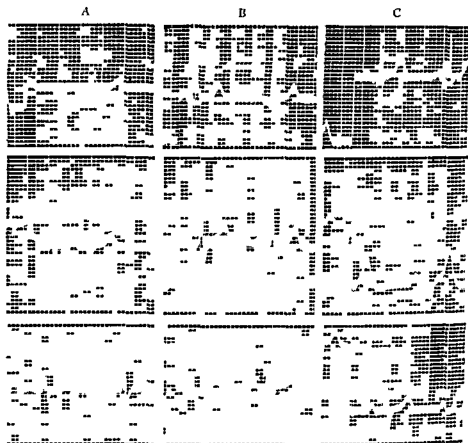



FIG 177 Electrocardiograms of the same patient showing variations produced by differences in string tension One millivolt is introduced in each instance A String too tight The deflection is only 0 5 cm B String tension correct One mv deflects the string one cm C String too loose The millivolt deflects the string 1 5 cm

The patient should be allowed to lie down and relax for a short time before the tracing is taken and the bed or table should be comfortable Conversa tions with the technician while the record is being taken should be avoided These points are all essential for the production of good electrocardiograms

The mental reactions of the patient may often cause variations in the skin current with the production of artefacts in the base line Movements of the patient during the examination or even the tension of the muscles resulting from an uncomfortable position naturally affect the electro-

cardiogram (Fig 176) Electric appliances in the vicinity may cause fuzziness of the string shadow although interference of this type can be eliminated satisfactorily in the modern instruments Quite marked artefacts in the record may occur if the skin resistance is too high because of haste on the part of the technician in preparing the patient for the examination This may result in over shooting of the deflection when one millivolt is introduced If the electrocardiogram is taken before this is corrected the waves will be too large and after each quick deflection the string will overshoot the zero level If the standardization is not exact the voltage of the waves will be incorrect (Fig 177) When electrocardiograms are taken with the patient in recumbency all subsequent records used for comparative study should be taken in a similar position since change in position produces alterations in the form of the electrocardiogram (Fig 178)

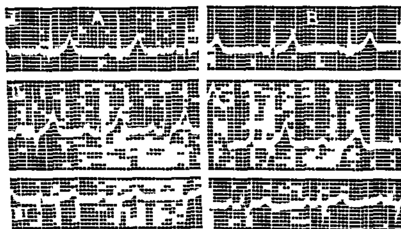


Fig 178 Electrocardiograms taken on the same person showing the effect of change in position A patient sitting upright in a chair B patient recumbent

## The P Wave

The P wave is the first wave of a group of deflections that makes up the electrocardiogram It precedes the QRS group and represents auricular activity Normally the P wave is a round upright deflection measuring not more than 0 1 second and averaging 1 to 2 mm in height In lead 3 of the electrocardiogram the P wave may be upright isoelectric (buried in and not projecting above the base line) or inverted (projecting below the base line) (Fig 179) If the rate of the heart is rapid the P wave may fall upon the downstroke of the preceding T wave In severe tachycardia the P wave may be lost within the T wave making the reading of the tracing difficult The P wave reappears however with the onset of a slower cardiac rate

## THE P R (P Q) INTERVAL

One of the most important measurements in electrocardiography is the time interval (measured on the base line) from the beginning of the P wave to the initial deflection of the QRS complex This is known as the P R interval and indicates the time consumed in the passage of the impulse from the S A node to the ventricular muscle It averages 0 15 second in the normal adult and should not exceed 0 20 second It varies with body size and cardiac rate With increase in the cardiac rate, there is a slight decrease in the P R interval

## THE QRS OR VENTRICULAR COMPLEXES

The Q R and S waves of the electrocardiogram represent ventricular activity The first deflection is downward and is known as the Q wave It is often absent in normal individuals The next deflection is upward and is known as the R wave The descending limb may dip below the base line forming an S-wave The exact form of these waves in each lead is determined by the direction that the excitation wave pursues through the ventricular muscle In some cases where several waves of low voltage compose the QRS group it is referred to as a W shaped or an M shaped QRS complex A difference in the direction of the waves of the QRS group may be caused by a change in the heart s position pregnancy an abdominal tumor high diaphragm or ascites The height of the QRS is generally between 5 and 20 mm (0 5 to 2 0 mv ) If over 20 mm the QRS group is spoken of as showing high voltage if below 5 mm it is said to have low voltage The duration of the QRS group should not exceed 0 1 second However in the presence of cardiac hypertrophy a longer time will be necessary for the impulse to pass through the thickened heart wall consequently the QRS interval is apt to be prolonged slightly The QRS may normally show notching or low voltage or both in lead 3

## THE T-WAVE

The T wave represents the retreat of negativity from the ventricular muscle Normally it is upright and measures between 0 15 and 0 5 millivolt and should not exceed 0 25 second in duration Significant and important changes in the T wave will be described later in relation to a number of cardiac conditions The T wave in lead 3 may be normally flat or inverted and occasionally, under some circumstances the T waves in leads 2 and 3 may show a similar alteration in normal individuals Such changes in $T_1$ are usually indicative of cardiac damage The T wave amplitude may be high in children following exercise in thyrotoxicosis or for no apparent reason In older patients the T wave amplitude tends to decrease

### The Q T Interval

The Q T interval is measured from the beginning of the QRS complex to the end of the T wave and represents ventricular systole It varies with the cardiac rate showing increase with slow cardiac rates and decrease with a rapid rate While there is still much to be learned about the im portance of this measurement it should always be studied carefully, espe cially in patients who have evidence of myocardial infections or chemical imbalance

### The RS T Interval

The RS T interval is the measured distance between the last QRS de flection and the upstroke of the T wave It is important to note whether this segment is raised above or depressed below the base line Distances exceeding 0 1 millivolt in either direction are abnormal The average dura tion of the RS T interval is from 0 24 to 0 28 second

### Alterations in the Waves of the Electrocardiogram and Their Significance

#### P wave changes

When the P wave is notched inverted or higher than 2 5 mm it may be said to be abnormal In mitral and pulmonary stenosis structural al terations are observed in the auricular musculature and these may be reflected in the P wave of the electrocardiogram Notching and increase in the duration of the P wave beyond 0 1 second are common changes in mitral disease Care must be used however in interpreting slight altera tions If the notching of the P wave is slight and occurs in leads 2 and 3 the finding should not be stressed If the notching is marked and occurs in lead 1 it is significant A normal upright P wave appears in the electrocardiogram when the impulse originates in the sinus node Should the impulse for cardiac contraction arise in any area outside the sinus node or pacemaker we may expect to find an inversion or alteration in size and shape of the P wave The P waves of auricular premature con tractions for this reason are often quite different from the P waves of the rest of the tracing The alteration in form of the P wave is usually proportional to the distance of the ectopic focus from the pacemaker (Fig 179 G) When a part of the P wave appears above the base line and a part below this level it is said to be diphasic (Fig 179 E) Diphasic or inverted P waves also occur in paroxysmal tachycardias of auricular origin Here the abnormal focus of impulse formation instead of initiating a single contraction for a time becomes the pacemaker and sends out im pulses at a rate usually exceeding 160 per minute Other parts of the conduction system may become more excitable and initiate impulses for cardiac contraction In such a case the impulse may spread backward (retrogression) and produce auricular contraction This may occur before

during or after the ventricular contraction, depending on the site of formation of the new impulse If auricular contraction follows that of the ventricle the P wave will appear following the QRS instead of in the usual location, and it will usually show inversion As we will see later there are no P-waves when auricular fibrillation is present Occasionally the sinus node may fail to generate an impulse or the auricles do not respond to the sinus impulse for the space of one or more cardiac cycles this is known as sinus arrest The P wave and consequently all the other waves of the cycle are absent (Fig 180 A and B)

ABNORMAL P R INTERVAL

Prolongation of the P R interval over 0 20 second is distinctly abnormal and points to auriculo ventricular block (Fig 181) It may accompany

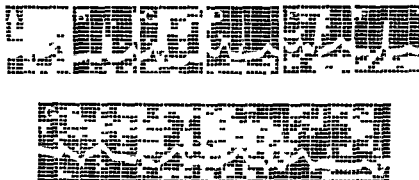


Fig 179 Abnormalities of the P Wave A normal B flat C pointed D widened, E notched and widened F inverted G a change is seen from upright to inverted caused by shift in site of impulse formation

diphtheritic carditis and is one of the most valuable signs in the presence of acute myocarditis Over 25 per cent of patients show prolongation of the P R interval during active stages of a rheumatic infection In many of these cases an overactivity of the vagus may be a contributing factor Increase in the P-R interval may be caused by defective conduction following interference with the blood supply to the bundle of His Consequently arteriosclerotic (rarely syphilitic) heart disease may produce this alteration

CHANGES IN THE QRS COMPLEX

It is important to remember that an impulse for cardiac contraction arising anywhere above the bundle will result in the normal spread of the excitation wave and produce a QRS complex of normal configuration Occasionally the distribution will be abnormal and a slight difference in the QRS complex results these beats are called aberrant

When we examine the QRS complex the following points must be noted

1/10 h Sec d    1/5 h ad 1/ 5 h S d    Mill m er

L ad N 1—B w en R gh and L ft A m

C

L ad N 2—B twern Right A m and Left L g

L ad N 3—B we Left A m and Left L g

FIG 180 Sinus arrest In A and B note omission of one complete card ac cycle In C the e are two complete cycles om tted from each lead (See Case No 66 )

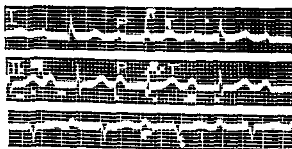


FIG 181 Prolongat on of the P R Interval or first stage heart block In lead the P R Intervals measure 0 3 second (See Case No 36 )

the direction of the major deflection (particularly in leads 1 and 3) the duration the configuration and the height Notching in lead 3 alone cannot be considered as an abnormal finding However, notching in leads 1 and 2 (particularly the M and W shaped complexes) must be considered as evidence of myocardial disease A slight notching seen near the base line is not as important as that observed at the top of the R wave (Fig 182) All low voltage QRS groups occasionally show notching in the absence of myocardial damage Marked notching of the QRS is seen in premature beats and in bundle branch block These departures from the

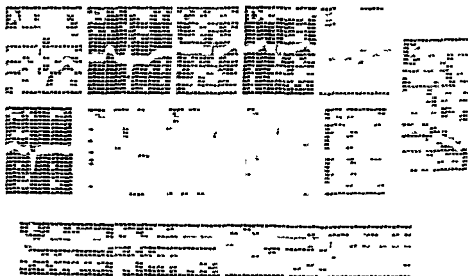


FIG 182 Variations in configuration of the QRS group A normal B diphasic C low voltage D notched (W shaped complex) E diphasic and slurred F shaded with deep S wave G slightly widened, notched and low voltage H slurred with deep S wave I widened and notched (bundle branch block) J deep Q wave in lead 3 K high voltage L respiratory variations in QRS amplitude in lead 3

normal will be considered in detail later Low voltage of the QRS sometimes occurs in patients with extensive edema hypothyroidism constrictive pericarditis and myocardial weakness The latter is the explanation of the low voltage electrocardiogram that often follows occlusion of the coronary arteries Low voltage QRS may be present occasionally in the absence of heart disease Consequently a low voltage electrocardiogram *can be interpreted correctly only in the light of the clinical findings*

The duration of the QRS group should not be more than 0 1 second when it exceeds this myocardial disease should be suspected unless one of the usual types of bundle branch block is present Normally the QRS is tallest in lead 2 consequently increased amplitude in this lead as a single finding has no significance The direction of the QRS groups in leads 1 and 3 is important in determining the axis deviation (page 597)

CHANGES IN THE RS T INTERVAL

Changes in this area between the end of the QRS and the beginning of the T wave have considerable clinical significance Elevation or depression of this segment in relation to the base line should always be carefully noted Digitalis may cause a marked alteration in this area Characteristic deformities also occur in the presence of coronary occlusion Both will be considered in detail later

CHANGES IN THE Q-T INTERVAL

The distance between the first part of the QRS and the end of the T wave can be said to correspond approximately to ventricular systole

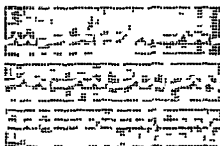


FIG 183 Prolongation of the Q T Intervals in hypocalcemia

and is lengthened in hypocalcemia (Fig 183) and after emergence from diabetic coma

CHANGES IN THE Q-WAVE

Recently a deep Q wave in lead 3 particularly when accompanied by a Q wave in lead 2 and an inversion of $T_3$ has been shown to be a

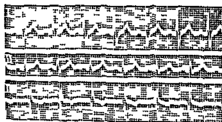


FIG 184 Deep Q wave in lead 3 In this instance it accompanied pregnancy and disappeared after delivery

significant electrocardiographic finding It often follows a posterior coronary occlusion On the other hand a deep $Q_3$ may accompany pregnancy (because of the high diaphragm) and disappear following delivery

(Fig 184) Here again correct interpretation of the electrocardiogram depends on a knowledge of the clinical findings If the history and physical examination are negative too much significance should not be attached to the isolated finding of a deep Q wave in lead 3 However, it always warrants a further study of the patient (see Figs 237 243 247)

T WAVE CHANGES

Inversion of the T wave particularly in lead 1 is significant The shape of the T wave and type of deformity in each instance are the important



FIG 185 Variations in the T wave A normal B large amplitude C diphasic D flat E inverted F deeply inverted (coronary type with deep Q)

factor For example it will be readily seen that the type of the T wave inversion differs in Fig 185 E and F from that observed in Fig 250 In the change of the T wave from its normal upright position to inversion a stage in the development is flattening Consequently flattening of the T-wave is an important observation particularly when it occurs in lead 1 However in some hearts particularly of the long ptotic or drop type too much significance should not be attached to this change If the T wave in lead 1 is low with upright and normal $T_2$ and $T_3$ in a person of this build it should be considered normal This again demonstrates that the decision regarding the exact meaning of an electrocardiographic alteration can be made only when the result of a complete examination of the patient is known This is particularly true in cases on inversion of the T wave in lead 3 It has been said that a slight degree of inversion of $T_3$ is normal Deep inversion however should arouse suspicion since this abnormality may follow a posterior coronary occlusion (Fig 185 F) This electrocardiographic finding particularly if accompanied by a deep $Q_3$ and inverted $T_3$ should direct attention to the past history of these patients and inquiry should be made concerning the presence of chest pain slight or severe in degree at some previous time

THE ELECTRICAL AXIS AND ITS DEVIATION

Change in the electrical axis of the electrocardiogram is important The axis may deviate to the right or to the left Although a complete study of axis deviation may prove complex even to the initiated, the main principles should be readily grasped Let us suppose that the solid arrow A B in Fig 186 A represents the value and direction of the various action currents generated in the heart as it contracts If lines are drawn perpendicular to the three sides of an equilateral triangle CED (representing the three leads of the electrocardiogram), the values of $a_1b_1$, a b and $a_3b_3$ will represent the values of the QRS in each lead (Fig 186 B) It

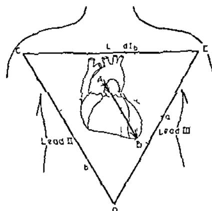



FIG 186 A Einthoven's triangle See text for explanation

will be readily seen that if the heart changes position in the chest and rotates to the right and assumes the position of the dotted arrow in the diagram the value of $a_1b_1$ will decrease When the arrow representing the action curve is exactly perpendicular to the base of the triangle the QRS voltage will be zero Should the rotation of the heart take place still further to the right the tip of the arrow will move toward the side CD and the current represented by the projections of the new position of the heart will flow in the opposite direction—and the QRS in lead 1 will now be inverted instead of upright Right axis deviation is then said to be present (Fig 187 A) A similar rotation of the heart to the left will bring the arrow A B perpendicular to the side DE In this position the value of the QRS in lead 3 will be zero Should the displacement be more marked and the point of the arrow move farther to the left the direction of the current as plotted on the side DE will be reversed Left axis deviation will then be present (Fig 187 B)

**Rule** Left axis deviation is present when the major deflection is upward in lead 1 and downward in lead 3 Right axis devia

tion is present when the major deflection of the QRS is downward in lead 1 and upward in lead 3 When the QRS complexes

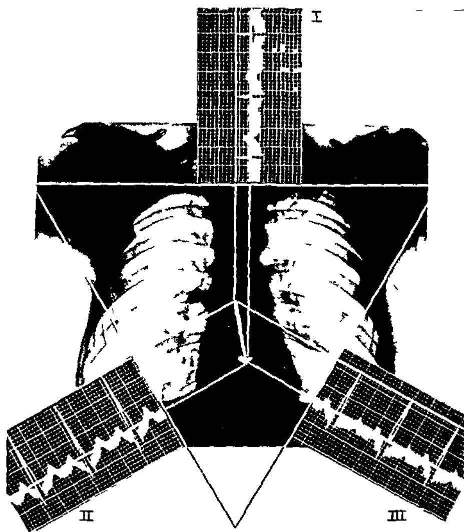



Fig 186 B Illustrates diagrammatically the effect of a transverse position of the heart on the size and direction of the main deflections of the electrocardiogram An Einthoven (equilateral) triangle is drawn about the heart The arrow represents both the electrical and anatomical axes of the heart The size and direction of the R wave in each lead is obtained by vertical projection of the ends of the arrow on that lead (From Master A M The Electrocardiogram and X Ray Configuration of the Heart Courtesy Lea and Febiger Co Phila )

are upright in leads 1 and 3 no axis deviation is present With no axis deviation QRS is higher than either $QRS_1$ or $QRS_3$ With either right or left axis deviation, it is lower

Axis deviation is generally interpreted as meaning preponderance of the right or left side of the heart Consequently in clinical conditions affecting the left side of the heart, *such as hypertension aortic regurgitation*

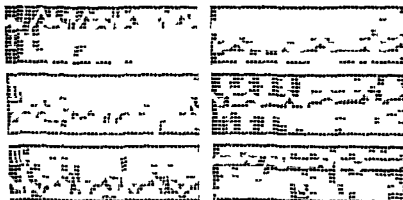


*Fig 187 A Right axis deviation B Left axis deviation*

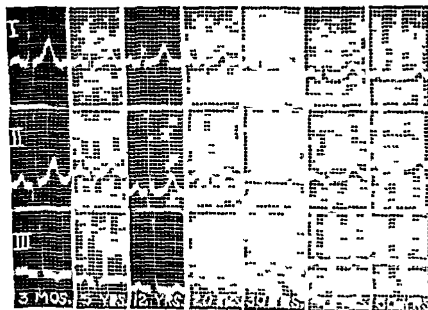



Fig 188 Normal electrocardiograms showing variations at different age periods

or aortic stenosis left axis deviation is commonly encountered Similarly in mitral stenosis with enlargement of the right ventricle some degree of right axis deviation may be expected in the electrocardiogram It can also be seen that IF EXTREME HYPERTROPHY OF THE HEART IS PRESENT AF

FECTING THE TWO SIDES EQUALLY WITH THE INITIAL RATIO UNDISTURBED THE ELECTRICAL FORCES WILL BALANCE AND NO AXIS DEVIATION WILL RESULT

Outside influences changing the position of the heart in the thorax may produce axis deviation A high diaphragm resulting from pregnancy or abdominal tumor or a large pleural effusion in the right chest may rotate the heart to the left and produce a left axis deviation Occasionally as the relationship between the mass of the left ventricle and the right ventricle changes in normal hearts, an axis deviation may result For example at birth and for the first few weeks of life, the mass of the right ventricle ex

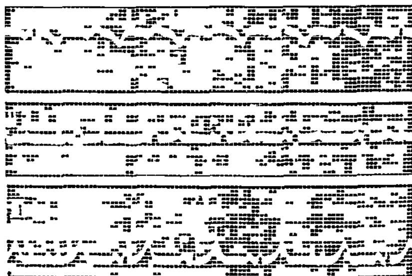


FIG 189 The electrocardiogram in dextrocardia Note inversion of all waves in lead I

ceeds that of the left and right axis deviation is seen (Fig 188) In the older age groups the relationship is in favor of the left ventricle consequently left axis deviation may appear as a normal finding

If we view the heart under the fluoroscope we will note that in some cases there is a marked change in its position following deep inspiration This amount of alteration in cardiac position can cause a shift in the electrical axis of sufficient degree to be recorded in the electrocardiogram (see Fig 182 L) If the respiratory excursion is slight or if the heart rests lightly on the diaphragm as it does in ptotic individual the change in cardiac position will be scarcely noticeable and there will be no alteration in the tracing

Congenital dextrocardia presents a characteristic electrocardiographic picture (Fig 189) All the waves in lead I are inverted This is what we would expect since the heart in these patients is opposite in its relationship to the leads A similar electrocardiographic picture results if the lead cords to the patient s arms are reversed by a careless technician

## READING THE ELECTROCARDIOGRAM

Before a detailed interpretation of the electrocardiogram is attempted a resume of all the clinical data available at the time of the examination should be in the hands of the physician giving the report If this rule is followed in every case many errors in the clinical application of electrocardiography will be avoided A suitable form covering all essential details is shown in Table XXIV Before the tracing is mounted for study make certain that the standardization is correct i e a string deflection of 1 cm follows the introduction of one millivolt potential difference (see Fig 165 C and D) Suitable seven inch strips should then be selected from each lead for mounting either in the elaborate (and more expensive) forms issued by the instrument companies or on a sheet of ordinary paper the size of the ward chart

TABLE XXIV

HOSPITAL OF
THE WOMAN S MEDICAL COLLEGE OF PENNSYLVANIA
DEPARTMENT OF CARDIOLOGY

REQUEST FOR 1—ELECTROCARDIOGRAM
2—ORTHODIAGRAM Check examination
3—CLINICAL OPINION desired

(Note Requests for electrocardiograms should be made in all cases where the clinical condition warrants and should be signed for by the chief of the service)

NAME Ward Out Patient Semi Private Private
Age (Please check status)
Date Service of
Blood Pressure Height Weight
RÉSUMÉ OF THE CLINICAL FINDINGS (Include your impression of the etiology heart size and rhythm)

CLINICAL DIAGNOSIS
HAS PATIENT RECENTLY RECEIVED DIGITALIS MORPHINE OR QUINIDINE? IF SO HOW LONG AND IN WHAT QUANTITIES?
ADDITIONAL LABORATORY DATA (Include here urinalysis blood count blood chemistry and serology)

________________________ M D

NOTE It is most important that above form be completely filled out in order that proper interpretation of laboratory data can be made With frequent requests (serial studies) only details of progress and additional laboratory studies need be furnished

In order that all the features of the electrocardiogram may be included in the description it is well for the beginner to adopt a definite method of procedure and follow it in all subsequent studies Important items are then not likely to be overlooked First note the rate of the heart This may be determined by placing a six inch ruler (usually equivalent to 30 of the fifth of a second divisions) beneath the strip of film and counting the number of auricular and ventricular cycles included in this measurement In Fig 190 the rate is 80 normal sinus rhythm is present and each auricular

wave (P) is followed by a ventricular complex (QRS) The rhythm is carefully studied by noting the presence of P waves their position in the cycle and the distance between them With a pair of architect's dividers span the distance between the peaks of the QRS complexes In Fig 190 it will be seen that they are equidistant The rhythm therefore, is regular, and the P waves show that it is of sinus node origin Next note the size shape and duration of the P wave in each lead In Fig 190 $P_1$ is widened measuring 0 12 second In lead 2 the P-waves are peaked, while in lead 3 they appear to be diphasic The P R intervals (measuring from the

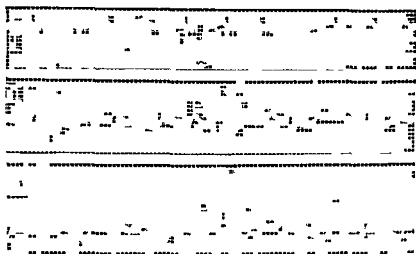


FIG 190 Electrocardiogram of a woman of 34 admitted to the hospital because of increasing dyspnea edema and hemoptysis She gave a history of two attacks of rheumatic fever in childhood Examination showed cardiac enlargement presystolic and diastolic apical murmurs and an accentuated pulmonic second sound Tablets of the whole leaf of digitalis 0 1 Gm (gr 1½) had been given after meals for three days prior to admission See text for reading

beginning of the P wave to the first deflection of the QRS) are 0 20 second in duration in lead 2 There is present a deep S wave in lead 1 The QRS in lead 2 has an R wave and a small S wave and is diphasic *The T wave in lead 1 is upright and normal In lead 2 there is a depres*sion of the S-T interval The same is present to a more marked extent in lead 3 Right axis deviation is seen (inverted QRS in lead 1 and upright in lead 3)

**Summary** Prominent and widened P or auricular waves, right axis deviation and depression of the S T intervals in leads 2 and 3

**Clinical Conclusions** The P wave changes and the right axis deviation support the diagnosis of mitral stenosis and suggest the presence of hypertrophy and disease of the auricular muscle and right sided cardiac enlargement The depressed S T intervals in leads 2 and 3 show beginning digitalis action

## SINUS MECHANISMS

If impulses arise from the sinus node at a rate of 60 to 100 per minute the P waves will have a normal shape and the cycles in the electrocardiogram will be equally spaced This is spoken of as normal sinus rhythm

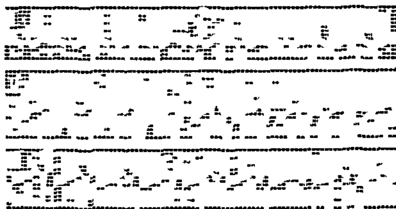


Fig 191 Electrocardiogram of a woman of 40 who showed all the clinical signs of thyrotoxicosis The basal metabolic rate was plus 55 per cent Rate 150 Sinus tachycardia

If the sinus node initiates impulses at a rate exceeding 100 per minute sinus tachycardia is present These impulses all follow the normal pathway A glance at the tracing (Fig 191 lead 1) shows that all the waves of the electrocardiogram (P QRS and T) have a normal relationship The in-

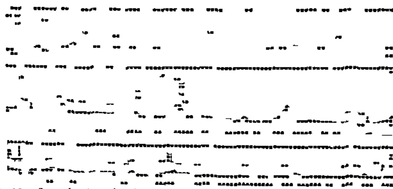


Fig 192 Sinus bradycardia (Rate 58) Note that the intervals between the T wave and the following P wave are prolonged

terval between the end of the T wave and the next P wave is shortened In lead 2 this T P interval is so decreased that the P wave arises from the downstroke of the preceding T wave

When the rate of impulse production is less than 60 per minute sinus bradycardia is present In Fig 192 the cardiac rate is 58 All the waves of the electrocardiogram appear to be normal, but the distance between the end of the T wave and the next P-wave (ventricular diastole) is prolonged Note the low voltage and notching of QRS in lead 3 THIS IS NOT AN ABNORMAL FINDING WHEN IT OCCURS IN LEAD 3 ALONE No abnormalities could be detected in the cardiovascular system of this patient The tracing was taken during convalescence from influenza

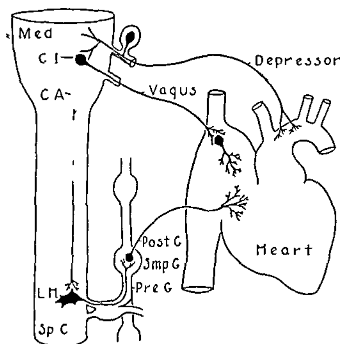



FIG 193 Diagram illustrating innervation of the heart

Although the initiation and transmission of the cardiac impulses do not depend upon the nervous system *the activity of the heart is controlled by extrinsic nerves* The vagus and sympathetic (Fig 193) are regulators of cardiac action increasing and decreasing cardiac rate according to the bodily needs of the moment The vagus sends fibers to the auricle and slows the heart through its influence on the sinus node and auricular muscle In youthful subjects who have normal hearts there may be noted a slowing down and a speeding up of cardiac rate during inspiration and expiration Inspiration speeds the rate while expiration slows the rate This irregularity is known as juvenile arrhythmia sinus arrhythmia or vagal arrhythmia and is caused by alterations in the vagal tone In some instances where the respiratory relationship is absent the arrhythmia may be caused by digitalis Figure 194 shows a marked sinus

arrhythmia Note that the individual waves of the electrocardiogram bear the same relationship to one another in all leads However the space between the T waves and the next P waves (T P interval) varies with each beat The remaining features of this record are normal

Very rarely the sinus node may fail to generate an impulse for contraction (see Fig 180 A) The result is a dead string and one beat of the heart is entirely blotted out The pause here is usually equal to two cardiac cycles Administration of atropine blocks the terminal branches of the vagus and tends to abolish the arrhythmia Figure 180 A is the tracing of a healthy young athlete who came to the hospital for study because the school physician detected dropped beats on auscultation Sinus arrest

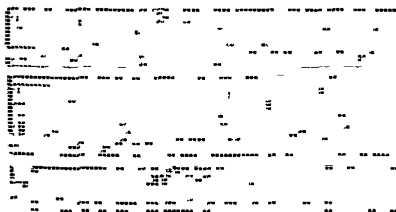


FIG 194 Sinus arrhythmia A slowing occurs during expiration and a quickening during inspiration

or standstill of the entire heart for one or two beats is caused by either failure of the sinus node to initiate the contraction impulse or the block of this impulse before it reaches the auricular muscle Its presence may be suspected clinically when a dropped beat is noted by auscultation However its true character can be recognized only when an electrocardiographic examination is made Vagal arrhythmias of this type are not unusual in well trained athletes

In Fig 180 B the same condition is seen Here however, it accompanies a deeply inverted T wave in lead 1 This patient had cardiac enlargement of the hypertensive type and a history of two episodes of prolonged chest pain following coronary occlusions In this case the sinus arrest was undoubtedly caused by a deficiency of the blood supply to the myocardial area containing the pacemaker

Very rarely two beats may be omitted The tracing shown in Fig 180 C was taken the day after an attack of chest pain lasting an hour in a man of 50 who had a previous history of hypertension The patient was sent to the hospital as a case of posterior coronary occlusion inasmuch as the

sudden fall in the pulse rate closely following the attacks suggested complete heart block Since the blood supply to the bundle arises from the posterior coronary artery, an involvement at this site was suspected However only a very slight increase in the P R intervals was noted The sinus arrest disappeared in a few days and the patient made an uneventful recovery

In Fig 195 an interesting variation will be noted After the third auricular beat a block of the impulse for contraction occurs at the sinus node In this instance the long pause is not interrupted by the occurrence of a P wave A slightly abnormal ventricular complex first appears This shows an important cardiac protective mechanism When the impulse for contraction failed to arrive at the A V nodal tissues in a time far exceeding the usual diastolic pause, the A V nodal structures in the emergency assumed the role of pacemaker and generated the impulse for contraction This phenomenon is known as VENTRICULAR ESCAPE Note the slightly different form of the ventricular complex in this cycle and the absence before it of the P wave of auricular contraction Both auricles and



FIG 195 Ventricular escape See text for explanation

ventricles in this instance contracted together Consequently the P wave is buried in the QRS The next impulse for contraction comes from the usual site in the sinus node as is shown by the reappearance of the normal P wave While ventricular escape is of no clinical importance it demonstrates the fine adjustment of the cardiac mechanism in the event of failure of the usual impulse for contraction It is likewise another example of the value of the electrocardiograph in explaining the cause of an abnormality of cardiac rhythm

Vagal action may also cause a shift in the pacemaker or site of impulse formation in the sinus node or it may cause the pacemaker to move down to the A V node for the space of a few beats A close study of Fig 196 reveals in addition to the bradycardia a variation in the duration of the P R intervals At times the P wave is buried in the QRS (first beat of lead 1) or it may be seen budding from the upright limb of the QRS (second beat in lead 2) Here the pacemaker is shifting or wandering back and forth between its usual site and the A V node THERE IS LITTLE OR NO CHANGE IN THE FORM OF THE QRS GROUPS WHEN THE IMPULSE FOR CONTRACTION ARISES ABOVE THE A V NODE Wandering pacemaker is again one of the finer points in electrocardiographic diagnosis and usually has no clinical significance

When the site of origin of the impulse shifts to the nodal tissues and

remains there A V nodal rhythm is said to be present Figure 197 shows this mechanism Note the absence of P waves the regular rhythm and the slow ventricular rate[4] The P waves are buried in the QRS groups This

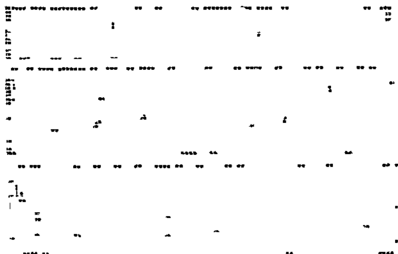


FIG 196 Wandering pacemaker Note the variations in the P R intervals in all leads as the site of impulse formation shifts from the sinus node to sections of the A V node

rhythm was recorded during the course of digitalization of a patient of 75 years of age who was suffering from arteriosclerotic heart disease and congestive failure In this instance it was a very important electrocardio-

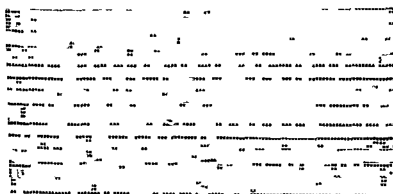


FIG 197 A V Nodal Rhythm Impulse a e from the A V node consequently auricle and ventr cle contract together and the I wave s bur ed in the QRS Rate 45

graphic finding The Heart Station called the ward to stop the digitalis since nodal rhythm often appears as a sign of beginning toxic action of this drug (page 8)

## PREMATURE CONTRACTIONS

### (Extrasystoles)

The ability to generate impulses for contraction is not a property possessed by the conduction system alone Any part of the heart structure may independently initiate these impulses and the electrocardiogram will show the presence of an isolated beat unrelated to the existing rhythm Since these beats arise from an abnormal focus they are spoken of as ectopic They arise before the next beat is due and consequently are also referred to as premature (The older textbooks called them "extrasystoles") These premature beats generally possess characteristic features that permit us

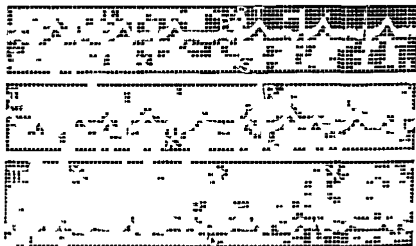


Fig 198 Premature auricular contractions In leads 2 and 3 the regular rhythm is interrupted at the sites marked x by premature beats from an abnormal auricular focus The P waves are smaller and the P R Intervals are shorter The site of origin of the beats is therefore low down in the auricle near the A V node

to place the abnormal focus for impulse formation in the auricle the A V node the bundle of His or the ventricles

If the premature contraction interrupts regular cardiac rhythm it is *usually followed by a long pause known as the compensatory pause* The first impulse arising from the sinus node following the premature beat is unable to initiate a contraction because of the refractory state of the heart muscle Consequently the whole heart pauses until the next impulse arrives from the S A node The unusually vigorous contraction that occurs after the premature beat is often felt and interpreted by the patient as "skipping" "a turn-over" or a "pounding" of the heart

Occasionally a premature beat may occur between two normal beats and not interrupt the dominant rhythm These are rare and are known as interpolated beats (see Fig 201 A) They are the only true extrasystoles

In the electrocardiogram the shapes of the premature beats arising in the same lead from identical foci are similar In the same lead differences in shape of two premature contractions from the ventricle point to two different foci

### Premature Auricular Contractions

Auricular premature contractions occur before the normal beat is due and are characterized by alteration in the P waves If the impulse arises in an auricular area outside the S A node the P wave shows a difference

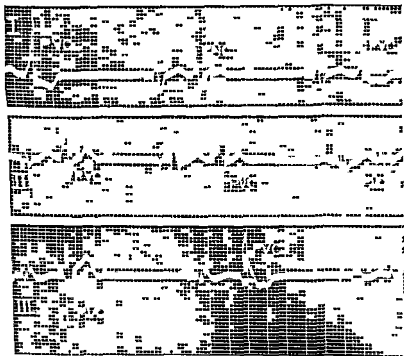


Fig 199 Nod l and ventricular premature contractions The premature beats are marked

in its shape In lead 2 of Fig 198 the third beat has a P wave of different shape since its point of origin is low down in the auricular musculature near the A V node Inasmuch as the distance to the nodal tissue is now shorter the P R interval is decreased In G of Fig 179 two premature beats of auricular origin are seen to occur together Their abnormal site of origin is indicated by the inversion of the P wave in each instance The QRS complexes of the auricular premature beats are similar to the others in the lead since the impulse descends to the ventricles along the normal conduction pathways Occasionally however the

ventricular beat may be slightly different in contour when the period of rest has been insufficient In some instances auricular premature beats may bring to light an early conduction defect For example the P R interval following a premature beat may be unduly prolonged and exceed the P R interval of the normal beats in this case we assume that some pathologic process is responsible for the tissue changes contributing to this delayed conduction

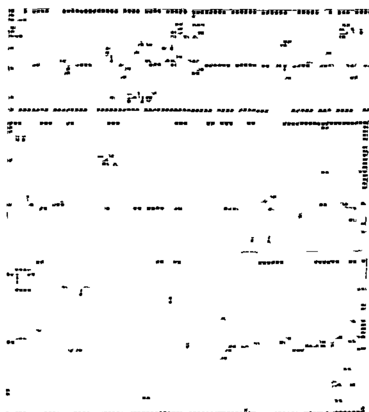


FIG 00 Premature ventricular contractions from foci in both ventricles

If the focus for the premature beat is in the A V node the auricles and ventricles often contract together because of the spread of the impulse in both directions (Fig 199, lead 1 beat 3) In this event the P wave may be buried in the QRS although in some instances it may follow the ventricular complex Occasionally the impulse reaches the auricle first and the auricular contraction precedes the ventricular In this instance however the P R interval will be much shorter than that observed in normal beats If the site of origin of the premature beat is very low in the A V nodal tissue the configuration of the QRS groups that follow may be slightly aberrant (Fig 199 lead 2 beat 2 See also Fig 195 beat 3)

Where auricular contraction follows an impulse traveling in a reverse direction along the specialized tissues we speak of this phenomenon as retrograde conduction

## Premature Ventricular Contractions

Premature ventricular contractions occur most frequently and are the usual cause of skipped beats They are readily recognized in the electrocardiogram (Figs 199 200 201) An impulse arising in the muscle of either ventricle causes premature contraction of both ventricles but the chambers do not contract together since it takes longer for the impulse to traverse nonspecialized muscular tissue For this reason the ventricu

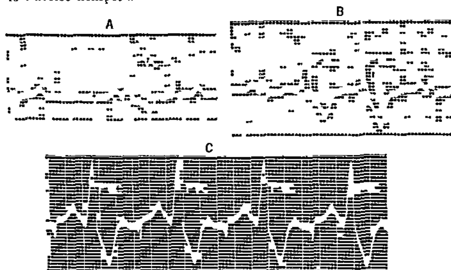



Fig 201 P ematu e ventr cular contractions A s ngle or isolat d B in pair The same irr t ble focu e l in t o impulses for co tract on C prematur beat follo ing each normal beat

lar complexes of the premature beats are wider than normal beats The irregular spread of the impulse often causes notching and the complexes are not preceded by P waves The rhythmic production of stimuli by the S A node is unaffected After the ventricles respond to the premature beat they are in a refractory state when the next impulse arrives from the pace maker Consequently there is a pause until the succeeding impulse re establishes normal rhythm

Definite localization at the present time is not possible Ventricular premature beats may occur as single beats (Fig 201 A) less often they appear in pairs (Fig 201 B) Occasionally they may follow each normal beat (Fig 201 C) particularly when they appear as a manifestation of the toxic action of digitalis It is usually stated that showers of premature beats from a variety of foci in auricles or ventricles or both are more apt

to occur in the presence of cardiac damage However, I have seen many exceptions to this rule

## THE PAROXYSMAL TACHYCARDIAS

Premature beats from any site may occur in short runs because of the fact that the ectopic focus takes complete command of the cardiac rhythm



FIG 202 A short paroxysm of ventricular tachycardia The onset and offset are seen Similar paroxysms in this patient terminated in paroxysms of ventricular fibrillation For this reason this irregularity is referred to as the prefibrillary type of ventricular tachycardia

and sends in a succession of stimuli (Fig 202) We may refer to this event as either a series of premature ventricular contractions or a short *run of tachycardia If the focus is in the auricle, we speak of the condi*tion as paroxysmal auricular tachycardia (Fig 203) Similar paroxysms may have their origin in an A V nodal focus (A V nodal paroxysmal tachycardia) (Fig 204)

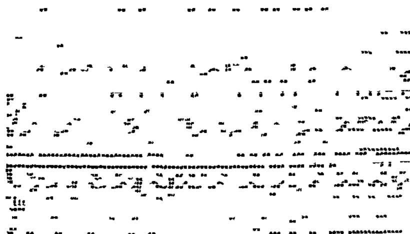


FIG 203 Paroxysmal auricular tachycardia

Clinically these paroxysms are characterized by the abruptness of their onset and offset and occur in patients who have no other sign of a cardiac abnormality as well as in those who present definite evidence of heart disease In most instances it is remarkable how well the heart muscle stands the strain of these abnormal seizures The complete absence of any of the usual signs of failure speaks well for the functional integrity of the myocardium in such cases Of course in older people who have degenerative changes the

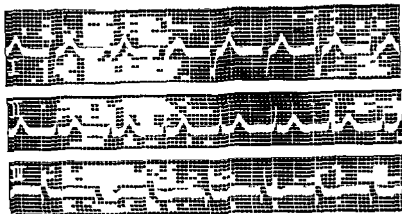


Fig 04 Paroxysmal nodal tachycardia

(A)

B

Fig 05 A Three leads of a tracing taken during a paroxysm of auricular tachycardia
B Paroxysm abolished by pressure over the right carotid sinus (applied at x)

sudden onset of a paroxysm of tachycardia may be followed quickly by evidences of cardiac failure or acute pulmonary edema The situation is then a medical emergency

In Fig 205 A we see three leads of a paroxysm of auricular tachycardia In B of this figure a tracing of lead 2 was taken while pressure was made over the right carotid sinus There is prompt return of sinus rhythm at the point marked 'X' Figure 206 shows a similar paroxysm in another patient, also stopped by carotid sinus pressure

Paroxysmal ventricular tachycardia (Fig 207) is a much more serious irregularity and occurs in the presence of organic heart disease The rate

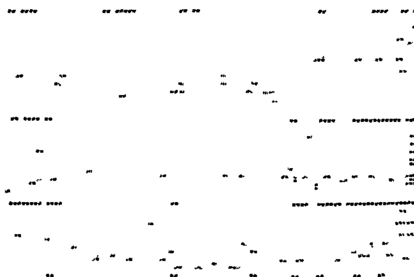


Fig 206 Three leads of an electrocardiogram showing the onset and offset of a paroxysm of auricular tachycardia The onset followed the second beat in lead 3 Note the inversion of the P wave in this lead indicating abnormal auricular focus The paroxysm was terminated at point marked 'x' by carotid sinus pressure

is usually slower than that of auricular or nodal tachycardias and varies from 130 to 170 beats per minute The tracing has the appearance of a series of premature ventricular contractions This is not unusual since a paroxysm of ventricular tachycardia arises from an ectopic focus in the ventricular muscle The QRS groups in these paroxysms are widened and usually notched

Paroxysmal ventricular tachycardia is often irregular while the other varieties are perfectly regular The two main causes of paroxysmal ventricular tachycardia are myocardial infarction and the administration of excessive doses of digitalis to a patient who has a badly damaged myocardium

The prefibrillary type of ventricular tachycardia is a dangerous as well as an imperfectly understood mechanism (see Fig 214 A) It is dangerous

because of the frequency with which it terminates in ventricular fibrillation Some observers believe that this arrhythmia is caused by a circus movement in the ventricle similar to the circus movement described by Lewis as occurring in the auricles They refer to it therefore as ventricular flutter

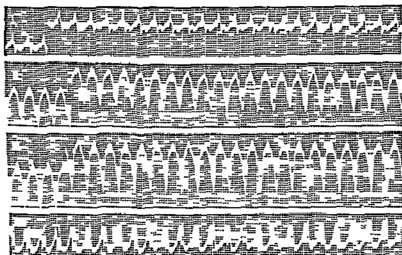


Fig 207 Ventricular paroxysmal tachycardia The onset followed an attack of acute coronary occlusion It was abolished by quinidine

In ventricular fibrillation the appearance of the tracing is characteristic (Fig 208) The waves show a disorderly arrangement and vary in size shape and duration from cycle to cycle There are only about 17 instances in the literature of recovery from ventricular fibrillation that are supported by electrocardiographic evidence Ventricular fibrillation no doubt



Fig 208 Ventricular fibrillation Note irregular coarse type of curve that varies from beat to beat This tracing was obtained during an Adams Stokes seizure in a patient suffering from hypertensive cardiovascular disease complicated by complete heart block

occurs more often than it is recorded I have seen two cases both of which were associated with varying degrees of A V heart block (see Chapter 12)

## HEART BLOCK

The pathway followed by the excitation wave from the auricle to the ventricular muscle has already been described Disease processes affecting

the bundle of His may delay the passage of this impulse, or if more extensive, may block it altogether The function of conduction in the bundle may likewise be depressed by an overactive vagus but rarely to the stage of complete heart block When myocardial invasion takes place particu

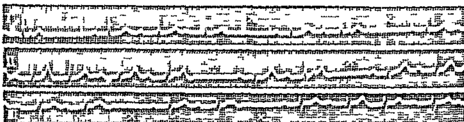


Fig 209 Incomplete heart block Dropped beats occur at sites marked "x" Note gradual increase in conduction time until beat is dropped (Wenckebach phenomenon)

larly of the diphtheritic type, the first hint of mischief may be indicated by a prolongation of the P R interval beyond the normal limit of 0 20 second (see Fig 181) If the lesion progresses the bundle may show an increasing inability to conduct impulses In this event the P R intervals gradually lengthen until a stage is reached where there is a total failure of the

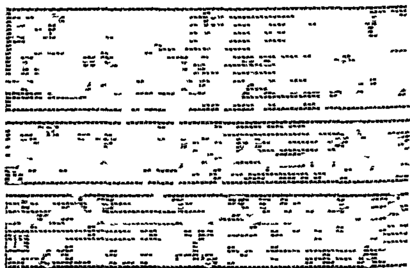


Fig 210 Heart block In this record every second beat is dropped

impulse to traverse the bundle (Fig 209) A beat will then be dropped out With this short rest, the bundle may be able to conduct the next impulse and a shorter P R interval appears The same process is then repeated until another beat is dropped This gradual increase in the duration of the P-R interval that ends in the total failure of the bundle to conduct

an impulse is known as the Wenckebach phenomenon The dropped beat is characterized clinically by an interruption of the pulse at the wrist and precordial silence The latter distinguishes between the pause of heart block and the pause following premature beats The extra sound produced by the premature beat may be readily ausculted The first stage of heart block eludes detection however and the electrocardiogram must be depended upon to make the diagnosis

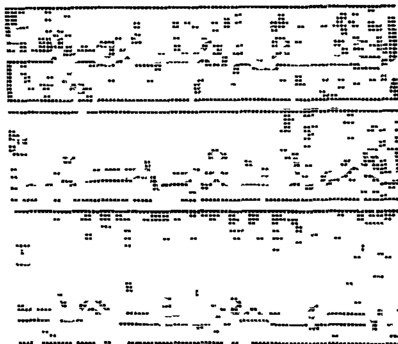


Fig 11 Complete h art block Auricula rate 76 Ventricular rate 38 The s nus node continues to control aur cular activity but the ventricles beat in response to a p cemaker that in tiates impulses for contract on at a slov er rate below the site of the les on

With progress in the lesion the blocked impulses may occur after each second beat (Fig 210) This is known as two to one heart block The ventricular rate is exactly half the auricular rate Complete dissociation or complete heart block between auricles and ventricles next occurs (Fig 211) and in order to survive the ventricles must establish a new center for impulse production below the level of the lesion This is exactly what happens the new center generally forming in the auriculoventricular tissues above the branching of the bundle The ventricular beats that are produced by stimuli from this site at a rate of 30 to 40 per minute show slight differences when compared to normal beats Often the disease process may extend down the conduction system to a lower level and the new

center may arise in one of the bundle branches The ventricular complexes are then widened and resemble those obtained in block of the bundle branch

Patients with heart block may be subject to Adams Stokes attacks at the time when complete heart block occurs The electrocardiograph in recent years has shed considerable light on the mechanism of cardiac action during and following these seizures Our laboratory has succeeded in taking tracings of three patients during Adams Stokes seizures Two of

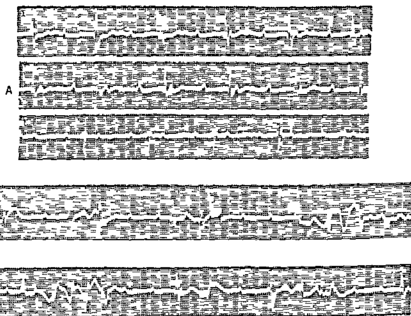



FIG 212 The electrocardiogram during Adams Stokes seizures For explanation see text

these patients developed attacks in the course of A-V heart block complicating hypertensive cardiovascular disease and the third following the onset of complete heart block that accompanied an acute (posterior) coronary occlusion The complete electrocardiographic records of cases 79 and 80 are included here with detailed interpretation

*FIG 212 A (Case 79) shows persisting complete heart block with auricular rate 88 and ventricular rate 44 The QRS complexes are notched and widened in all lead and left axis deviation is present Strips similar to B were observed to occur immediately prior to an onset of Adams Stokes seizure Here there are numerous premature beats occurring singly and at the end of the strip in pairs The auricular rate following the ectopic beats is more rapid than in the control averaging 107 beats per minute Strip C of Fig 212 is similar to B with the exception of the number of premature ventricular beats seen to occur in succession Short runs of three are present at the beginning and end of the strip

In Fig 213 Strips A B and the first half of C represent a continuous tracing taken *during an Adams Stokes seizure Strip A shows the prefibrillary type of ventricular tachy*

*This fine print can be omitted by the reader without destroying the continuity of the text

cardia with a rate of ?10 per minute Some slight irregularities are present in thi strip but on the whole all of the widened complexes conform to the same pattern In strip B from X1 to X there i a change in the configuration of the ventricular complexes they become v idened and differ in shape from beat to beat The last part near X is quite typical of ventricular fibrillation From the point X? to the end of the strip B we again note the presence of th prefibrillary type of ventricular tachycardia This arrhythm a continues throughout the first part of strip C At the middle of C a strip of tracing showing an arrhythmia similar to A and C is omitted Thirty seconds later the second part of strip C was obtained th s sho s a typical attack of ventricular fibrillation Note the presence in this strip of auricular beats Strip D is continuous

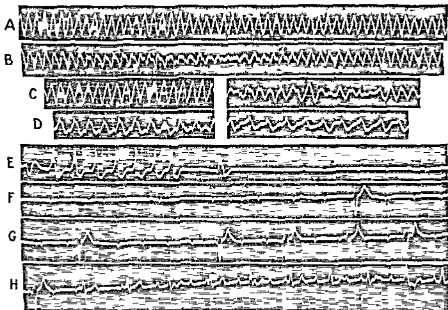



Fig 13 Electroc rdiogr am ecorded dur ng Adams-Stok s se zure See text for expla nat on

with C and sho vs emergence f om an attack of ventricular fibr llat on The QRS groups are widen d nd slurred and there appears to be a P wave b fore each QRS group Th v dth of the QRS complexes dec ases to ard the end of the str p The last half of trip D taken a minute later shov biza re configu at n of the QRS groups with S T interval depress on Ten attacks were reco d d showing electroca diographi find ngs sim lar to those d scr bed above

The next attack i sho n in strips E to H of Fig 13 this i a continuous record and is ncluded to sho v a d fferent type of emergence In strip E the QRS complexes are markedly aberrant v ith deep S T int rvals The rate of these complexes grad ally d creases to vard the end of the str p The e is a slight pause toward the m ddl of the strip and this s followed by an ectop beat which init ate a per od of ventricular standstill lasting 16 8 se ond Au icular be ts are seen at fir t coming through at a very slov rate and then grad ally inc easing in frequency A ventr cul ectop c beat is s en to vard the end of the str p th s niti tes a return of th v nt cular beats whi h occ r first at cycle length of 4 6 and then 4 2 and 2 0 seconds The rate of the ventr cular beats gradually increases until at the beg nn ng of str p H the rate ap

proaches 50 per minute The auricular rate in this strip is also rapid averaging 125 per minute Looking back on the auricular beats in strips F and F they will be seen to vary in shape This is probably because of the fact that the beats arise from different foci in the auricular muscle

Fig 214 shows additional interesting features of some of the shorter paroxysms that were recorded Strips A and B are continuous At the start of strip A there are five successive ventricular premature beats followed by a pause The next ventricular premature beat initiates a short paroxysm of the prefibrillary type of ventricular tachycardia The shape and the amplitude of the ventricular complexes of this paroxysm show a gradual change The last cycles are quite aberrant Strip B shows complete A V heart

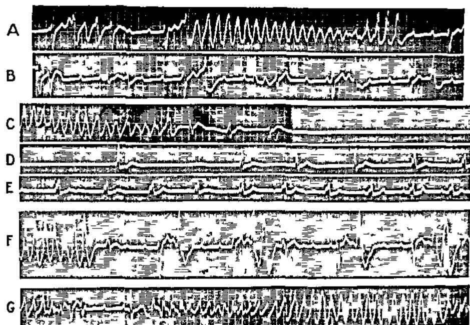



FIG 214 Tracings recorded during shorter seizures in same patient See text for explanation

block interrupted by numerous premature beats from various foci C D and E are continuous strips In the first part of C we note a paroxysm of prefibrillary type of ventricular tachycardia This ends with the appearance of an idioventricular rhythm the rate of which is 52 per minute The idioventricular center however apparently fails for a period of 8 2 seconds during which time complete cardiac standstill is observed The auricular beats are likewise absent in this strip The function of the idioventricular center gradually returns and occasional cycles are seen in strip D These recur with greater frequency until a rate of 46 per minute is reached in strip E The ventricular complexes however are considerably widened measuring 0 16 second There is complete absence of auricular beats from point Y in strip C to point Z in strip D In the beginning of strip E the auricular rate is slow measuring 40 per minute this gradually increases in frequency until at the end of strip E it approaches the rate of 100 per minute Strips F and G are a continuous tracing At the beginning of strip F there is a short paroxysm of the prefibrillary type of ventricular tachycardia terminating in a single ventricular ectopic beat Following this the idioventricular center continues to be interrupted by the occurrence of numerous ventricular premature

beats some in couples and others appearing in pairs Strip G shows several paroxysms of the prefibrillary ventricular tachycardia with ventricular complexes of different types

Fig 235 A shows a three lead control tracing of the type obtained on the patient whose history appears in Case 80 A two to one A V heart block with auricular rate of 80 and ventricular rate of 40 is present Note the *widening and notching* of the QRS complexes in leads 2 and 3 In strip B sinus rhythm is seen with prolongation

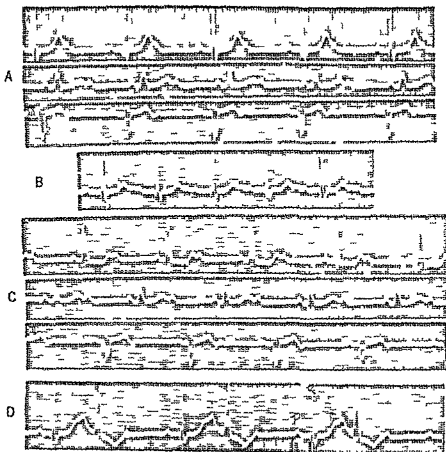


Fig 235 Control tracings (Case 80) taken before onset of Adams Stokes seizures For explanation see text

of the P R intervals to 0 3 second Strip C shows a two to one heart block with QRS complexes notched and widened in all leads Note the difference between QRS1 of this strip and QRS1 of strip A Strip D shows a two to one heart block with coupled ventricular premature beats from various foci This tracing preceded the paroxysm of prefibrillary type of ventricular tachycardia

In Fig 236 A to F is a continuous tracing obtained during a typical Adams Stokes seizure Strip A shows a prefibrillary type of ventricular tachycardia The ventricular rate of 300 per minute These complexes present a definite regularity in rate as well as in form although in different places in the lead a slight difference in

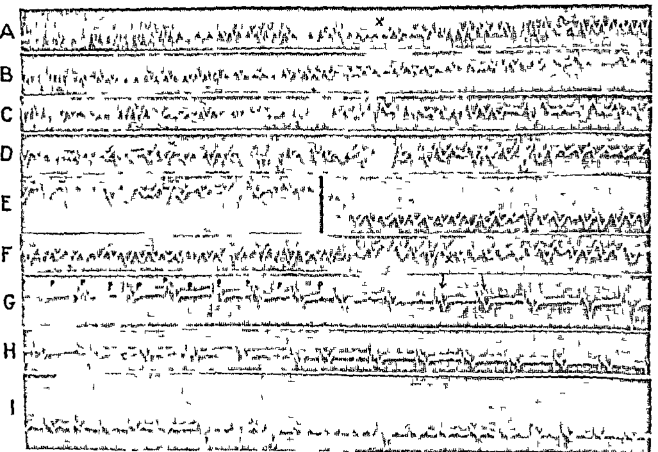



Fig 16 Long paroxysm of prefibrillary type of ventricular tachycardia terminating in *ventricular fibrillation* with recovery For detailed explanation see text

shape is noted For instance at point X sufficient irregularity exists to constitute a short paroxysm of ventricular fibrillation The first third of strip B is the same as A in th la t two third of this strip the ventricular complexes are s en to become more irregular than in th previous strip From the end of strip B and in luding strips C D E and F we see a continuous paroxysm of ventricular fibrillation Note the extreme irregularity in the siz amplitude and sequence of the ventr cular complexes In some places a semblance of regularity may be noted but this is present for an extremely brief period The total duration of thi paroxysm of ventricular fibrillation is 4 seconds Auricular beats can be noted in places during the above paroxysm Figure 16 (G to I) was made a fe v seconds after the end of the paroxysm and shows complete A V d ssoc ation v ith auricular rate of 140 and a ventricular rate of 64 Note th R T elevation in the initial cycles gradually decreasing to vard the end of the str p v here the R T segments are practically at the isoelectric line Here the auricular rate and the id oventricular rhythm are much more rapid than in th control tracing This is not infrequently observed immediately following emergence from a paroxysm th anox c state actin as a st mulant to the respective centers Note that the la t cycle of strip G is aberrant Strip H shows that the grade of heart block is nov tv o to one ind cating a return to more normal function across the A V nodal tis ues This is maintained to th end of the strip with a gradual increase in the rate of the idiov entr cular center Strip I shows a prolongation of the P R intervals to 0 3 second ith a one to one respon e Occasional ectopic beats are seen

The Adams-Stokes seizure recorded here was the only one that the patient had during her two v eeks stay n the hospital She was ambulatory on di charge A follow up letter at the end of five m nth found her in fairly good condition She reported no recurrence of the seizures

## BUNDLE BRANCH BLOCK

Various pathologic processes may cause block of the bundle on either side The appearance of the electrocardiogram obtained under these cir

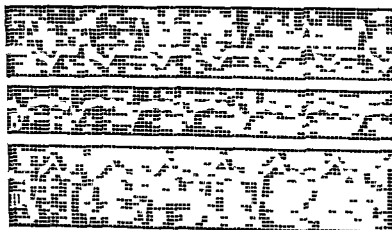


FIG 17 Bundle branch block Note the wid ned notched QRS complexes with the T v ave oppo ite n direction to the main complex This is common type of tra in It results from block of the left bundle branch

cumstances may be understood if we follow the happenings in the heart The ventricle on the side of the intact bundle branch contracts first The

impulse then travels through nonspecialized muscle tissue to the opposite ventricle where a delayed contraction occurs This is reflected in the appearance of the electrocardiogram The QRS group is widened because of the longer time consumed by the impulse in spreading through nonspecialized tissue of the muscle and notched because of the irregularity of this spread The T waves are usually opposite in direction to the main deflection of the QRS Increased voltage of the QRS is usually present To determine the site of the lesion apply the same rule as in axis devia



Fig 18 Right bundle branch block An isolated premature ventricular contraction is seen in lead ? This is uncommon type of bundle branch block

tion * For example in Fig 217 the QRS groups are upward in lead I and downward in lead 3 This is complete block of the left bundle branch (common type) In Fig 218 the QRS groups are down in lead 1 and up in lead 3 This is complete block of the right bundle branch At times we see the QRS groups widened and directed upward in all three leads Although previously referred to as arborization block or block in the Purkinje network these tracings are now regarded as partial block of the main bundle branches (Fig 219) In some cases of this type the bundle branch block may not be complete and a return to normal conduction may be seen every third or fourth beat (Fig 220)

* This is still a controversial question Some authors do not agree with the views presented here

When bundle branch block is seen we can usually be safe in suspecting myocardial damage There are however some exceptions to this rule When a bundle branch block is associated with a short P R interval (Fig 221) it should not be viewed as a sign of disease These patients

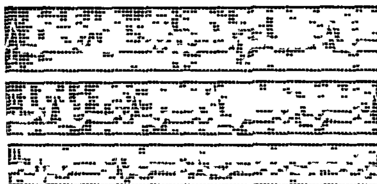


Fig 219 Intraventricular block

are often subject to attacks of paroxysmal rapid heart action To explain the occurrence of functional heart block of the bundle branch type it has been claimed that an additional conduction pathway (bundle of Kent)

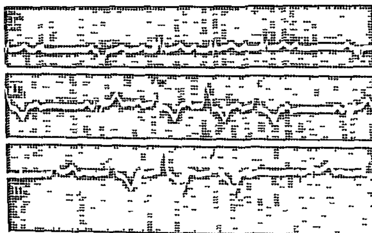


Fig 0 Transient bundle branch block

is present that transmits the impulse in a shorter time than it takes to traverse the usual pathway In some cases a series of sudden attacks of paroxysmal tachycardia arising from an auricular focus may severely tax the conduction system Notching of the QRS may be present and in some cases a widening of the QRS may be seen in normal individuals Here

again is an instance where the electrocardiogram must be interpreted entirely in the light of the clinical findings

A diagrammatic summary of the more common types of tracings associated with block at various levels in the conduction system is shown in Fig 222



Fig 221 Bundle branch block with short P R Intervals

## AURICULAR FLUTTER

The term auricular flutter has been advanced for an uncommon but interesting derangement of auricular function characterized by the establishment of an abnormal circulating contraction or flutter wave Flutter is supposedly produced by the continual passage of this wave around the auricular musculature near the entrance of the great veins (circus movement) Each time the wave passes, there is a radial spreading of the contraction impulse (*Fig 223*) When the electrocardiogram records this movement of the flutter wave, in a favorable lead the string will be seen to be in continual motion The auricular rates in cases of flutter are high averaging from 250 to 360 per minute Fortunately the ventricle even if healthy, could not respond to all the impulses arriving at the A V node Usually every second impulse penetrates and causes a ventricular contraction i e a two to one heart block is present Varying grades of block may appear consequently the pulse at the wrist in auricular flutter may be *120* to *170* When the slower ventricular rates are present the condition is often missed because it is unsuspected In flutter the heart rate is constant in all positions Pressure over the carotid sinus however, may be used to distinguish between paroxysmal tachycardia and flutter If flutter

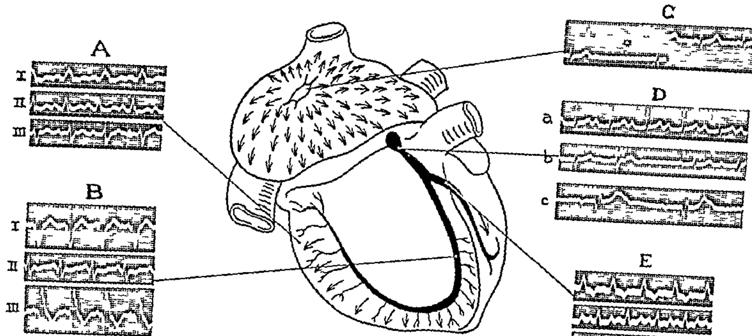



Fig 2? Diagram illustrating various types of b ock and th site of the lesion in the conduct on system in each instance A Int aventricular (ar borization) block B Right bundle branch blo k C S nus arrest (sino auricular block) D Heart block a I olongat on of P R Interval (first stage) b Dropped beats c Complete d ssociation E Left bundle branch block

is present carotid sinus pressure may cause a sudden halving of the pulse rate If this does not take place the pulse in flutter will usually become

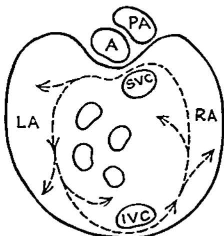



FIG 223 Diagram showing course of flutter wave in the auricle (Redrawn from Pardee) *

slower and often irregular Carotid sinus pressure in cases of flutter may even cause ventricular stand still while in paroxysmal tachycardia if the

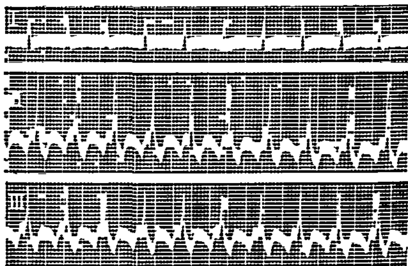


FIG 224 Auricular flutter The ventricle contracts following each second revolution of the circus wave (2:1 response)

attack is not stopped and normal rhythm restored no effect whatsoever will be noted on the rate or rhythm of the pulse If the patient is in re

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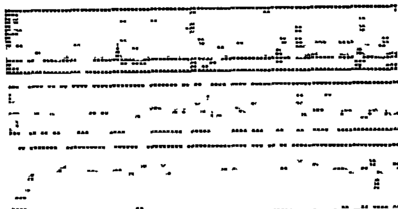


FIG 2 5 Au icular flutt r There is a ventricular response to every third flutter vave (3 1 response)

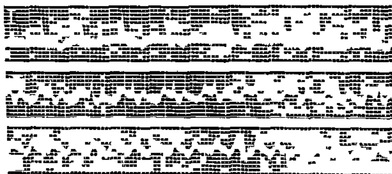


FIG 26 Aur cular flutter In lead 1 note the varying grees of ventricular re ponse

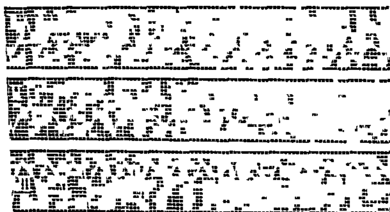


FIG 7 Au icular flutter 1 to 1 ventr cular response

cumbency in the proper light the neck veins may be distinctly visible in which event the very rapid flutter movements are often observed as they are transmitted back along the venous column from the auricle In many

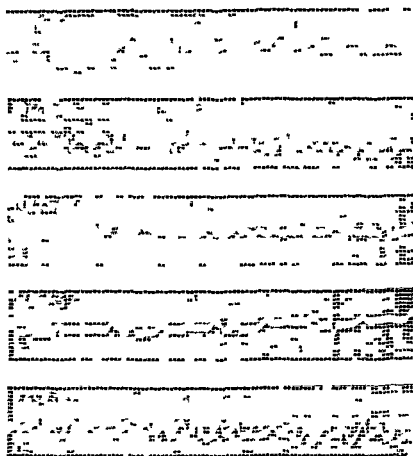


Fig 28 Series of electrocardiograms (lead 2) illustrating the effect of digitalis on auricular flutter A Normal rhythm B Auricular flutter Varying degrees of ventricular response C Following administration of digitalis The flutter has been converted into fibrillation D After withdrawal of digitalis normal rhythm appears E Recurrence of flutter two months later This patient had rheumatic heart disease cardiac enlargement and advanced mitral stenosis

cases although flutter may be suspected it cannot be diagnosed without an electrocardiogram

The appearance of the tracing in auricular flutter is characteristic (Fig 224) The flutter waves are generally seen to the best advantage in leads 2 and 3 where they appear as uniform up and down oscillations Closer inspection shows that the auricles as a whole appear to enjoy no period

of diastole for there is seen no beginning and no ending of the auricular movement The ventricular rate in flutter depends on the degree of A V block While a two-to-one block is usually present higher and even varying degrees of block are not uncommon (Figs 225, 226 228) A few cases where the ventricle has responded to every wave of the circus movement (one to-one response) are on record These are serious happenings particularly in diseased hearts since the strain on the myocardium is usually proportional to the increase in the ventricular rate The patient whose tracing appears in Fig 227 was a housewife of 52 who suffered from coronary disease and hypertension She died suddenly a few minutes after the onset of an attack of auricular flutter with a one to-one response This fatal attack was similar to the one recorded here

Digitalis is the drug of choice in flutter and should be given in full dosage to increase the grade of A V block Following digitalis a two-to-one response may be converted into a four to one The action of this drug may be watched by a series of electrocardiograms If the digitalis is continued fibrillation replaces the flutter and when the drug is withdrawn sinus rhythm usually returns (Fig 228) Although quinidine has been recommended digitalis is generally more successful in bringing relief to the patient with this type of circus movement and is the drug of choice Flutter commonly complicates thyrotoxic and rheumatic heart disease and may appear suddenly in patients suffering from arteriosclerotic heart disease It may occasionally appear in patients of any age who have apparently normal hearts (paroxysmal flutter) (page 391)

## AURICULAR FIBRILLATION

Auricular fibrillation is closely allied to auricular flutter and is by far the most important of the cardiac arrhythmias Here again it may be useful to recall the circus movement theory of Garrey in order to obtain a clear understanding of this mechanism In flutter the circus movement takes the same pathway through the auricular muscle at each revolution In fibrillation the course of the circus is wholly erratic (Fig 229) It weaves its way in and out of the auricular myocardium in an irregular fashion taking the course permitted by the physiologic state of the muscle at the moment Co-ordinated auricular contractions cease The circuits of fibrillation are completed at varying times but are al

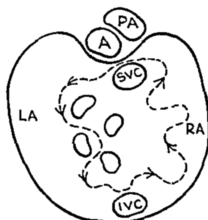


Fig 9 Diagram showing irregular course followed by the fibrillation waves in the auricular muscle

ways more rapid than in flutter Auricular fibrillary rates of 400 to 600 are not uncommon In consequence the ventricular response to the series of rapid irregular impulses arriving at the A V node is likewise irregular A typical electrocardiogram of fibrillation is shown in Fig 230 Note first the absence of the usual P waves that represent co-ordinated auricular contraction At times in place of the auricular waves a series of irregular 'f or fibrillation waves appear The QRS groups in auricular fibrillation are irregularly spaced and are of supraventricular type i e the origin of the beat is above the junctional or A V tissues

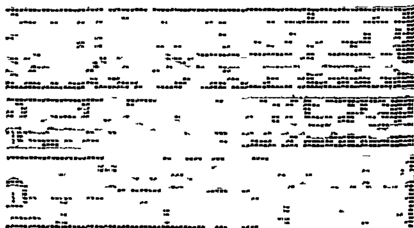


FIG 30 Auricular fibrillation Note total irregularity of the rhythm The P waves are replaced by f waves (seen best in lead 3 of this tracing) Note the depression of the S T intervals in leads 1 and caused by digitalis Left axis deviation is also present

The diagnosis of auricular fibrillation can nearly always be made clinically However if the ventricular rate is slow it may elude detection If it is possible to exercise the patient the distinction may be made at once since exercise increases the pulse irregularity in fibrillation At times premature beats occur with such frequency that the cardiac rhythm is entirely irregular Exercise abolishes the premature beats, and the rhythm of the heart becomes regular Premature beats may occur with auricular fibrillation but they arise always from a nodal or ventricular focus and never from a focus in the midst of such auricular turmoil

Auricular fibrillation may be permanent or paroxysmal The former is more common In rare cases the paroxysmal variety may be present and represent the only abnormality in the cardiac examination (page 595)

The f' or fibrillation waves may be quite prominent (Fig 230) especially when the arrhythmia complicates mitral stenosis and hypertrophy of the auricular muscle is present On the other hand in hearts with poor auricular myocardium, the 'f waves may be small or scarcely visible at all (Fig 231) In some leads the f waves may be so regular that they

suggest the presence of auricular flutter (Fig 228 C) The term impure flutter has been employed by some laboratories to designate this type of electrocardiogram and may be retained if preferred for these tracings probably represent a transition stage between flutter and fibrillation

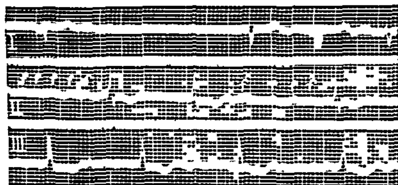


*Fig 31 Auricular fibrillation Note absence of P waves th total irregularity of rhythm The S-T Intervals are depressed in leads 2 and 3 Right axis deviation is present*

## CORONARY ARTERY DISEASE INCLUDING ACUTE MYOCARDIAL INFARCTION

Myocardial infarction that follows the occlusion of a coronary vessel produces characteristic alterations in the form of the electrocardiogram A study of these changes often enables the physician to state the site of the infarct while frequently repeated tracings or serial electrocardiograms are helpful in following the healing process

In recent years the contributions to this branch of electrocardiography have been many However a uniformity of opinion on all issues unfortunately does not prevail at this writing The terminology is likewise in an unsettled state While the recent standardization of the direct or precordial leads will ultimately be of great value at present much confusion exists regarding the interpretation of the tracings taken by the various technics In this brief sketch of electrocardiography I will try to avoid as much as possible the controversial points and briefly state the facts that appear at this time to be definitely established

At the start we must realize that the coronary arteries cannot influence the form of the electrocardiogram unless the blood flow through them is temporarily or permanently interrupted and the cardiac muscle is affected If serious disease of the walls of the coronary arteries is present but so situated that it does not interfere with the nourishment of the heart muscle no alterations appear For this reason an electrocardiographic diagnosis of uncomplicated coronary sclerosis is impossible However if a coronary artery is suddenly occluded with the production of an area of myocardial infarction marked changes usually appear sooner or later in the electro-

cardiogram in well over 90 per cent of the cases These changes first described by Pardee in 1920, take place in the RS T segment of one or more leads They consist of a downward, sharply peaked T wave (coronary T wave of Pardee) with an upward convexity of the RS T interval (Fig 232) *Following an acute infarction the RS T segment arises above the iso electric level (monophasic curve)* As healing progresses, the RS-T interval returns to the base line

The tracings obtained in the presence of an area of acute infarction usually fall into two groups Barnes and Whitten (1929) first designated

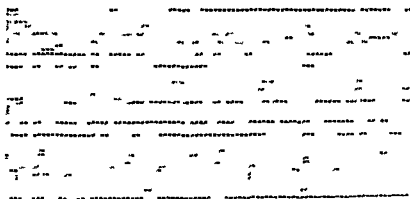


Fig 232 Electrocardiogram made 1[illegible] hours after an attack of severe chest pain in a man of 50 suffering from hypertensive cardiovascular disease Note the typical RS T interval elevation in lead 1 and the depression of the same segment in lead 3

these as the $T_1$ and $T_3$ types The $T_1$ pattern is characteristic of an infarction involving the anterior portion of the left ventricle and consists of an elevation of the RS-T segment in lead 1 and a depression of the RS T segment in lead 3 (Fig 232) Infarction of the posterior basal portion of the left ventricle produces an elevation of the RS T segment in lead 3 and a depression of the same interval in lead 1 (Fig 233)

Later investigations by Wilson (1933) first called attention to the importance of the initial deflection or Q wave in the diagnosis and localization of areas of infarction This investigator referred to the $Q_1$ and $Q_3$ types In the $Q_1$ type associated with an anterior infarction the Q wave appears and persists in lead 1 (Fig 234) while a posterior infarction may produce a deep Q wave in lead 3 (see Fig 237) It may be demonstrated by animal experimentation that curves of these types are routinely produced by an injury to the cardiac muscle of either the right or left ventricle [1]

Following an occlusion the area of injured muscle undergoes change from day to day This is reflected in the electrocardiogram but each patient will be found to differ in regard to the speed with which this variation takes place Characteristic alterations may appear in the RS T intervals 30 min

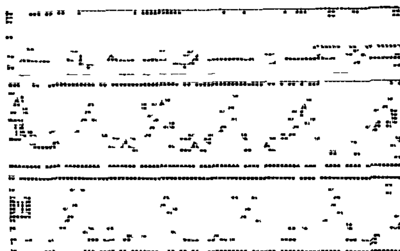


Fig 233 Tracing taken three hours after an attack of acute indigestion in a laborer of 40 Both father and mother suffered from coronary disease with anginal seizures Note the RS T interval alterations in leads 2 and 3 The descending limb of the QRS group does not return to the base line but continues into a large T wave (monophasic curve)

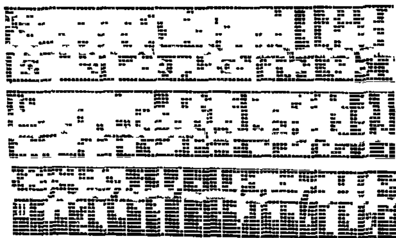


Fig 234 Evidence suggesting an old anterior coronary occlusion Note the Q wave in lead 1 the flat T and diphasic T Inversion of T and left axis deviation are also present

utes after the seizure or they may be delayed for several days For this reason serial studies are advisable whenever the clinical features are suggestive and the initial electrocardiogram is negative

While the electrocardiogram in coronary occlusion may change its form from day to day the alterations are more marked and more rapid during the first weeks following the accident When healing of the infarct is complete the indirect (limb) leads may show a return to the normal configuration Frequently however the T-waves will remain inverted, and their peculiar cove like shape should at once arouse suspicion concerning their mode of origin (see Fig 238) The clinical history should then be searched for proof At times RS T deformities may return to normal with healing of the infarct but the reduction of the blood supply to a vital spot like the bundle of His may result in delayed conduction and a permanent prolongation of the P R interval However, when the only remaining feature of the occlusion is deeply inverted $T_3$ care should be used in its interpretation since inversion of $T_3$ is not an uncommon finding in normal electrocardiograms If a deep $Q_3$ is present we are more certain of the meaning of the deep $T_3$ but in the absence of the Q wave in lead 3 the clinical history is the only guide in the interpretation Lead 2 may help in making the decision 'M or W shaped complexes in lead 2 as well as a deep $Q_3$ should always make us suspicious of an old posterior infarction (see Fig 182D)

Alterations in the auricular complex have also been observed to follow an occlusion of the coronary artery Characteristic changes in the P waves and the P R segment have been produced in animals by compression of the auricular arteries and are directly related to the myocardial ischemia Similar changes have been observed in man following coronary occlusion There may be depression of the P R segment with notching and inversion of the P wave Arrhythmias may occur with the displacement of the pace maker from the S A node

Electrocardiograms following exercise are occasionally of value in clarifying the diagnosis of angina pectoris Where an objective method is desirable in insurance examinations or when the history is typical but the patient neurotic the appearance of characteristic changes in the RS T intervals or T-waves of the electrocardiogram following exercise may be valuable The normal patient will show no alteration in the T waves after exertion nor will he show depression of the RS T intervals exceeding 1 mm below the base line The patient with angina may show depression or elevation of the S T interval of over 1 mm in leads 1 and 2 following exercise often associated with some T wave inversion However a negative result does not exclude the diagnosis of angina

Similar alterations in the RS T segment and T wave of the electrocardiogram have been produced by Levy and his co workers in patients suffering from coronary sclerosis by inducing anoxemia A special apparatus is used for this test by means of which the patient breathes a constant percentage mixture of oxygen Levy reports alterations in the T wave in amplitude

and occasionally in direction and depression of the RS T segments in the indirect leads This test may also be of great value when the diagnosis is in doubt

## PRECORDIAL LEADS

Studies of the usual three leads by various groups of workers soon demonstrated that coronary occlusion in some instances could occur in the absence of the characteristic electrocardiographic alterations just described

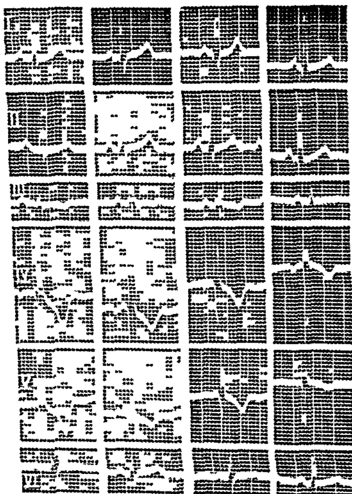


FIG 35 Series of normal six lead electrocardiograms taken by the old techn Lead 4—between apex beat and angle of left scapula; lead 5—between the apex beat and the left leg lead 6—between the angle of the left scapula and the left leg Note the deep Q waves in leads 4 and 5 and inverted T waves in the same leads

Consequently the re introduction by Wood and Wolferth[4 0] in 1931 of Waller s method (1887) of applying electrodes directly to the chest wall was a valuable advance that greatly increased the efficiency of the electrocardiographic method in the diagnosis of cardiac infarction These investigators recommended a fourth lead obtained by applying the right arm electrode over the apex beat and the left leg electrode directly opposite

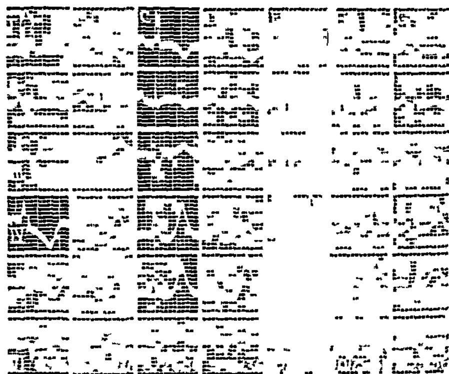


FIG 236 Serial tracings following attack of coronary occlusion (Case 40) The direct leads (4 5 and 6) were taken by the old technic In A the T waves in leads 4 and 5 are inverted In B they are upright Note the succession of changes in the T waves in lead 1 and from A to C In G the indirect leads are almost normal The alterations persist in the T waves of the direct leads

on the back of the chest at the level of the angle of the scapula In this original fourth lead (Fig 235) the first deflection was normally downward and was referred to as a Q wave the following upward deflection was termed an R wave and the last deflection normally directed downward was called the T wave

Various studies of the fourth lead soon showed that in some cases it exhibited the characteristic features of occlusion when the usual three leads were normal In other instances it was found that it aided considerably in establishing more firmly the diagnosis suggested by the limb leads In

the presence of acute infarction in the anterior portion of the left ventricle significant changes occur in the fourth lead (Fig 236) The Q wave disappears The S T segment is depressed Gradually $T_4$ becomes upright and increases in height The T wave may later return to normal but the Q wave in this lead does not reappear When $Q_4$ is absent in the direct lead obtained by the use of the old technic this finding should always suggest a previous anterior infarction even in the presence of normal indirect leads

A posterior infarction produces

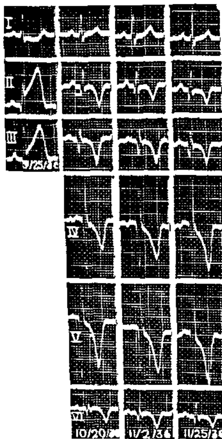



Fig 37

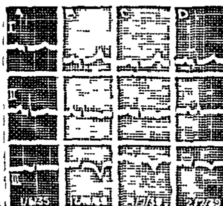



Fig 38

Fig 37 Serial tracings (old direct leads) following posterior coronary occlusion While T and T are deeply inverted the changes in the usual or indirect leads are characteristic and furnish sufficient evidence in this case

Fig 238 Serial tracings (Indirect leads) Patient an American woman of 55 who had no signs of cardiovascular disease on physical examination The blood pressure was 1 0/80 Urine negative Blood sugar normal A Electrocardiogram was made because of pain in the left elbow The pain was dull and unrelated to exertion Similar tracings were obtained over the course of three years B Taken one week after an attack of indigestion The pain was epigastric and lasted a few minutes The findings suggest an area of infarction on the posterior surface of the heart when compared to A C and D show gradual disappearance of the signs from the record If D was obtained and other records of this patient were not available a diagnosis of coronary occlusion could not be made

changes in the fourth lead that are usually transient and much less marked The RS T segment is elevated and the depth of the T wave diminishes

The Q wave remains These changes fade rapidly and subsequent tracings show Q and T waves of increased amplitude In my experience the standard leads reflect the changes of a posterior infarction much more clearly than lead 4 (Fig 237) Consequently while the precordial lead is a valuable aid in the diagnosis of various types of myocardial injury study of the usual indirect leads should not be neglected (Fig 238)

When the use of the fourth lead became popular, many laboratories, as the result of continued experimentation with a number of connections from various locations on the thorax adopted their own combinations As a result no unanimity of opinion existed as to where the electrodes should be placed The confused situation that resulted discouraged the practitioner's attempts to learn at this time the features of many of the different combinations of chest leads A standard method of applying electrodes was very much needed to clarify the situation To this end in 1938 a joint committee appointed by the American Heart Association and the Cardiac Society of Great Britain and Ireland considered the matter and made the following recommendations with reference to the routine use of a single precordial lead *[3]

1 It is recommended that those who employ a single precordial lead place the precordial electrode upon the extreme outer border of the apex beat as determined by palpation If the apex beat cannot be located satisfactorily by palpation the electrode may be placed in the fifth intercostal space just outsid the left border of cardiac dulness, or just outside the left midclavicular line if percussion of the heart is unsatisfactory Where precordial leads are taken by a technical assistant the position of the precordial electrode should be marked on the chest by the physician

2 It is recommended that a single precordial lead in which the precordial electrode has the location specified in the preceding paragraph be known as *Lead IV B* when this electrode is paired with an electrode in the left interscapular region *Lead IV R* when it is paired with an electrode on the right arm *Lead IV L* when it is paired with an electrode on the left arm *Lead IV F* when it is paired with an electrode on the left leg and *Lead IV T* when it is paired with a central terminal connected through equal resistances of 5 000 or more ohms to electrod s on each of the three extremities mentioned

It is suggested that for all ordinary purposes *Lead IV R* or *Lead IV F* be employed The latter lead should have the preference until it has been established that the former which is somewhat more convenient is equivalent to the latter for all practical purposes or yields results of equal value

3 It is recommended that in taking the precordial leads specified the galvanometer connections be made in such a way that relative positivity of the apical electrode is represented in the finished curve by an upward deflection (a deflection above the isopotential level) and relative negativity of the apical electrode by a downward deflection

It is urged that this convention be adhered to in case of precordial leads other than those specified and also in the case of all leads in which the one electrode is placed much closer to the heart than the other In other ord it shall be the standard convention in taking such leads to make the galvanometer connections in such a way that relative positivity of the electrode nearer the heart is represented by an upward deflection

4 It is recommended that with the galvanometer connections made as described in

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the preceding paragraph the deflections of precordial leads be designated by the symbols P Q R S and T and that in the application of these symbols the same conventions be employed as in the case of the standard limb leads

5 It is recommended that in taking precordial lead the electrocardiograph be so adjusted that a deflection of one centimeter in the finished record corresponds to a potential difference of one millivolt as in the case of the standard limb leads Any reduction in sensitivity made necessary by very large deflections should be clearly indicated on the curve preferably by photographing the effect of introducing a potential difference of one millivolt into the galvanometer circuit

6 It is recommended that the greatest dimension of the apical electrode employed in taking the leads specified in this report be 3 cm or less A circular electrode between cm and 3 cm in diameter should ordinarily be employed (see Fig 174)

7 It is recommended that the terms Lead IV (R F etc ) apical lead apex leg lead etc be used henceforth only in connection with the lead specified in this report

The standardization of the single precordial lead accomplished the American Heart Association has added a supplementary report for the workers who wish to employ multiple precordial leads A summary of the report follows

## Multiple Precordial Leads

When leads from two or more precordial points are employed it is suggested that the precordial electrode be paired either with an electrode on the left leg or with a central terminal connected through an equal resistance of 5000 or more ohms to electrodes on the right arm left arm and left leg It is suggested further that in the first case the letters CF followed by a subscript and in the second case the letter V followed by a subscript be employed to designate such leads

The position of the precordial electrode shall be indicated by the subscript used according to the following plan subscript 1 shall be used for the right margin of the sternum 2 for the left margin of the sternum 3 for a line midway between the left margin of the sternum and the left midclavicular line 4 for the left midclavicular line 5 for the anterior axillary line and 6 for the left midaxillary line When the letters and subscripts specified are employed it shall be understood that in the case of the sternal leads the precordial electrode has been placed in the fourth intercostal space and that in the case of the other leads it has been placed upon a line drawn from the left sternal margin in the fourth intercostal space to the outer border of the apex beat (or to a point at the junction of the midclavicular line and the fifth intercostal space) and continued around the left side of the chest at the level of the apex beat or of the junction mentioned

The advantages of making the galvanometer connections in such a way that relative positivity of the precordial electrode is represented in the finished curve by an upward deflection and relative negativity of this electrode by a downward deflection are as follows

1 This method makes it possible to assign the letters Q R and S to the individual deflections of the QRS group in exactly the same manner as in the case of the standard limb leads without violating the general principle that as far as possible deflections which have the same origin or the same significance should invariably bear the same name In particular it makes it possible always to assign the same letter (R) to the onset of the intrinsic deflection which signals the arrival of the impulse at the epicardial surface of the portion of the heart subjacent to the precordial electrode without departing from the customary method of labelling the QRS deflections

2 In cases of infarction of the anterior wall of the heart this method yields ventricular complexes characterized by abnormally large initial downward deflections (Q waves) and sharply inverted T wave of the cove plane of coronary type These complexes are practically identical with those which have long been considered

characteristic of myocardial infarction in the case of the standard leads and they may be described in the same terms

3 The P deflections and the T deflections are normally upright There are great advantages particularly from the standpoint of one who is teaching electrocardiography or of one who is beginning the study of this subject in a system which makes upright T waves invariably normal whatever the lead

4 The use of the terms plus and minus and of the symbols + and — greatly simplifies things In the case of the precordial leads one electrode the precordial electrode is of much more importance than the other In the discussion of the principles upon which the interpretation of the precordial electrocardiogram rests it is necessary to refer frequently to the potential of the precordial electrode and in connection therewith to employ the terms and symbols mentioned Since we are accustomed to speak of downward deflections as negative and to prefix measurements of such deflections as negative much confusion and misunderstanding will be avoided if the deflection of the tracing is upward when the potential of the precordial electrode is positive and downward when the potential of the electrode is negative



Fig 239 Electrocardiograms of three normal persons including the direct lead (4F) recommended for routine use Note normal variations

**Nomenclature** For the convenience of those who wish to make statistical studies of the QRS group to measure and tabulate the QRS deflections or to classify or characterize QRS deflections of different types it is imperative that the individual deflections of the QRS group be designated by distinct symbols even though the naming of these deflections may involve the application of rules more or less arbitrary

The adoption in the case of precordial leads of symbols different from those employed in the case of the standard leads might have some advantages This would however have at the same time tremendous disadvantages It would add an entirely new terminology to clinical electrocardiography which is already regarded by many as an abstruse and incomprehensible subject and would greatly increase the number of technical terms which beginners in this field would have to learn It would invite other attempts to improve upon electrocardiographic terminology and would stand little chance of prompt and universal acceptance The adoption of new symbols for the initial ventricular deflections would greatly complicate the use of such terms as the P R interval the QRS interval the RS T segment and the RS T displacement which could not then be logically used in reference to precordial leads For these reasons it was decided that the deflections of the precordial leads should be designated by the same letters as those of the standard limb lead

In addition to establishing uniformity in the connections for single or multiple precordial leads these recommendations make the interpretation of the tracings much easier since all the waves in the normal chest leads are upright This also permits the precordial leads to be correctly

designated by the same letters as those of the standard limb leads Lead 4F is the direct lead recommended for routine use (Figs 239 240 241)

While precordial leads display their greatest usefulness in the diagnosis of acute infarction, they are occasionally useful in the study of other con

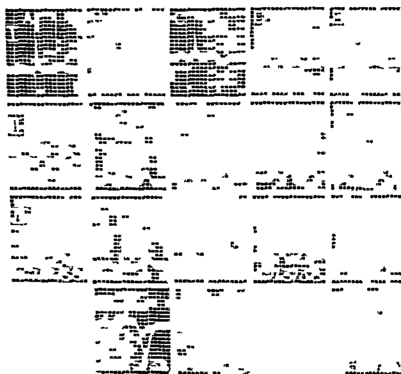


FIG 240 Value of direct lead Patient a foreman of 48 admitted to Receiving Ward of Woman s College Ho pital compla nin of abdominal pain He gave a history of stoma h trouble and ind estion for five ye r A tentat ve d agnosis of ruptured peptic ulcer w s m de A T ken on adm s ion Ind ect leads no mal B Taken six hours late Th direct lead 4F show change suggest ve of acute infarction (anterior) A slight alte at on may be noted in th RS T interval of the indirect lead C Six days later change n RS T interval n lead 1 more pro iounced D Two months later Evi d nce fad ng from indirect leads D Follow up exam nati n a year later shows no ev dence of presence of area of healed infarct on

ditions For example in the presence of puzzling auricular arrhythmias the P wave may be better visualized in the chest lead For this record the chest electrode is placed over the middle or just to the left of the sternum In making the differential diagnosis between acute coronary occlusion and pulmonary embolism chest leads are helpful while they permit a more complete study of myocardial injuries arising from other causes for ex ample trauma and pericarditis

## DIAGNOSIS OF CORONARY OCCLUSION BY THE RHYTHM OF THE ELECTROCARDIOGRAM

Conduction disturbances are not infrequently observed following acute coronary occlusions Since the bundle of His is supplied by a branch of the

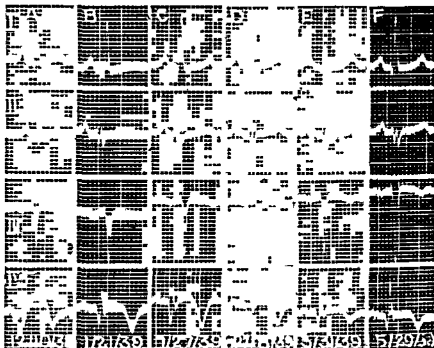


Fig 241 Value of direct lead Patient a salesman of 35 Blood pressure normal Family history positive for coronary disease and angina (both father and mother) A Taken three hours after onset of sharp pain in left shoulder Usual or indirect leads while suggesting the presence of heart disease ( w complex of lead ) failed to show *presence of area of infarction Lead 4F however taken at the same time* shows characteristic monophasic curve While the T waves in leads 1 and 2 of B are flat there is no additional evidence in the indirect leads of the series of the presence of infarct A series of characteristic changes may be seen in lead 4F

right coronary artery posterior occlusions may produce varying degrees of auriculoventricular block Occlusions may also result in block of a bundle branch The rich protective blood supply about the bundle of His and the re establishment of the circulation to this area by collateral flow following the occlusion probably account for the return of normal rhythm in Fig 242 The sudden appearance of a cardiac irregularity of this type with or without chest pain should always suggest the possibility of coronary occlusion The onset of the complete heart block in the patient whose tracing is shown in Fig 243 was attended only by faintness while the patient whose tracing appears in Fig 244 had Adams Stokes seizures

that occurred during the last six hours of life The signs of posterior coronary occlusion usually accompany those of heart block Note the alter

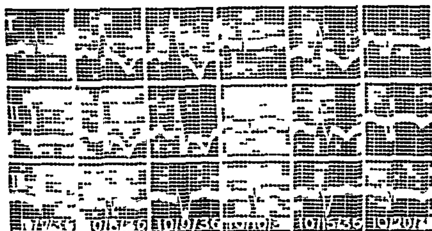


Fig 242 Serial electrocardiographic studies (usual leads) taken over the course of two weeks following an acute coronary occlusion Note the appearance of a bundle branch defect on some days and the return subsequently of normal conduction After four weeks of bed rest the characteristic widening of bundle branch block did not reappear

ation in the RS-T interval in leads 2 and 3 of Fig 243 At times a bundle branch defect may obscure the usual curves of infarction in both indirect and chest leads Consequently in these cases it is hazardous to base a diag

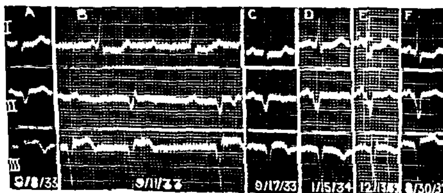



Fig 243 Transient complete heart block following posterior coronary occlusion See Case No 43

nosis on the electrocardiogram However if complete heart block appears suddenly and the clinical picture is suggestive the diagnosis of occlusion

should be suspected An acute coronary occlusion may also usher in a paroxysm of ventricular tachycardia (*Fig 245*)



Fig 244 Electrocardiogram made a few hours after the onset of attack of chest pain Note presence of complete heart block The patient had a series of typical Adams Stokes seizures and died six hours after this tracing was recorded

## ELECTROCARDIOGRAPHIC ALTERATIONS FOLLOWING DIGITALIS

As we would expect the electrocardiogram shows marked changes following the administration of digitalis Often these are characteristic of the action of the drug although at times they cannot be distinguished from

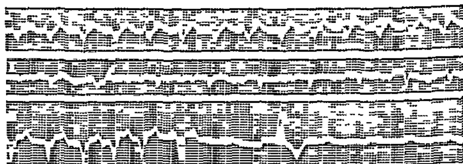


Fig 245 Paroxysmal ventricular tachycardia following onset of initial attack of coronary thrombosis in an Irish janitor of 51 Hypertension present for 10 years The patient died in the receiving ward while this tracing was being recorded

the effects produced by cardiac disease The alterations produced by digitalis in the electrocardiogram are helpful to the physician in directing therapy and may prove of great benefit to patients since electrocardio

graphic changes may serve as warnings of the approach of toxic action Death from overdosage of digitalis is not unknown consequently any laboratory aid in checking the effect of the administration of this drug is an advantage

**P and T Waves** Digitalis affects the heart rate and rhythm It may alter the shape of the P and T waves of the electrocardiogram When the influence of the vagus predominates in the early stages of digitalis action slowing of the heart may be present In toxic amounts digitalis may speed the heart with the production of paroxysms of ventricular tachycardia

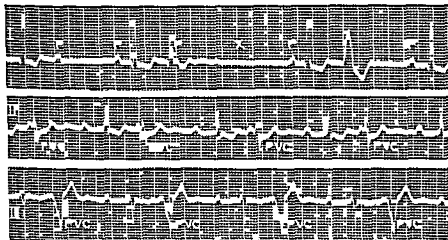


Fig 246 Electrocardiogram of a patient suffering from rheumatic heart disease mitral and aortic stenosis Eighteen grains of the whole leaf of digitalis were administered over the course of the week before above tracing was made Note premature ventricular contractions following normal beats the prolongation of the P R Intervals and the dropped beat at x

**A V dissociation** may be produced by digitalis with slowing of the heart (Fig 246) This is caused by vagal action and is a toxic manifestation Both flutter and fibrillation have been reported following massive doses of digitalis A V nodal rhythm may also appear (see Fig 197)

Varying degrees of heart block may be induced by digitalis when fibrillation of the auricles is present Here digitalis registers its most dramatic effects acting almost as a specific remedy in patients with high ventricular rates (Fig 247)

**Premature Beats** Digitalis frequently produces premature beats in diseased hearts Bigeminal rhythm is a characteristic sign of the toxic action of digitalis (Fig 248) Isolated premature beats should also be considered in the category of warning signs when digitalis is being administered When these are observed if the drug is not properly regulated serious paroxysms of ventricular tachycardia may appear However it must

be kept in mind that the presence of premature contractions alone is not a contraindication to digitalis therapy Many times following successful digitalization this arrhythmia entirely disappears

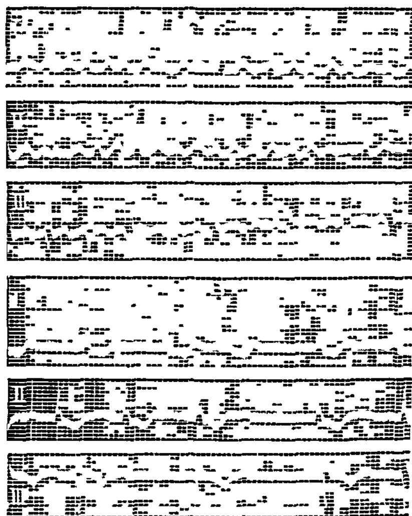


Fig 47 Two three lead electrocardiograms illustrating the effect of digitalis in a patient suffering from rheumatic heart disease and auricular fibrillation A Before digitalis Ventricular rate 170 B Same patient three days later following the administration of 1 3 grams (19½ grains) of whole leaf of digitalis Note the slowing of the ventricular rate in B (8 ) and depression of the S-T Intervals in all leads There was striking clinical improvement

Vagal action is probably responsible for the alteration in the P wave of the electrocardiogram that occurs following toxic doses of digitalis These

changes are encountered most frequently in lead 3 and include all the alterations in the P waves shown in Fig 179 They are however of little clinical significance

T Wave The greatest influence of digitalis is upon the T wave of the electrocardiogram If Figs 247 and 248 are closely inspected the change will be seen to be brought about by a depression of the S-T segment of the tracing This pulls the T wave down with it producing an appearance in the electrocardiogram that is quite typical of the action of digitalis In

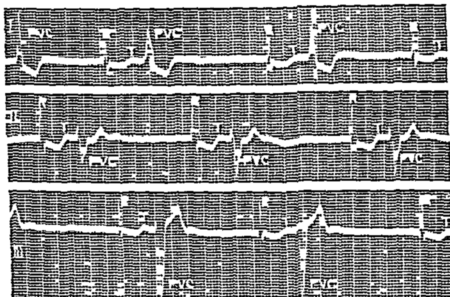


FIG 48 Effect of overdigitalization Pacemaker displaced to A V node (nodal rhythm) Note the premature ventricular contraction following each nodal beat and the depressed S T Intervals

patients with normal hearts and upright T-waves this electrocardiographic change follows the administration of digitalis It may also be observed in patients who have mild myocardial damage In badly damaged hearts however upright T waves may become inverted in a manner not at all characteristic At times the inversion may resemble the coronary type of T wave If the T wave is already inverted by disease the administration of digitalis may cause it to assume an upright position (Fig 249) Consequently interpretations of these changes may lead to false impressions unless the electrocardiographic laboratory is in possession of exact information concerning the amount of digitalis that the patient has received and the date the administration was begun It is likewise important to remember that the laboratory cannot always tell the stage of digitalization by an inspection of the shape of the T wave The change in the T wave reaches

its height while the action of digitalis is still a therapeutic one Continued dosage may produce toxic signs but causes no further change in the T wave

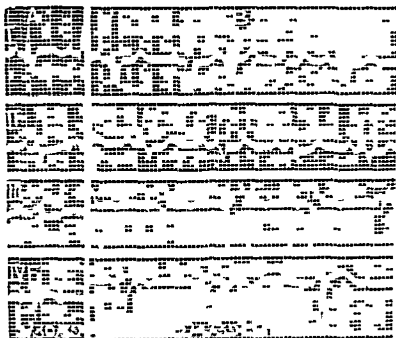


FIG 49 Effect of digitalis on T Wave A Tracing taken during an attack of congestive failure The patient was a woman of 48 suffering from hypertensive cardiovascular disease B Taken after the administration of 1 5 grams (? ½ grains) of the whole leaf of digitalis Note change in T and T In lead 1 of B there is an interpolated premature ventricular contraction There is a slight widening of the QRS groups

## ELECTROCARDIOGRAM IN CHILDREN

While the fundamentals of electrocardiography are essentially the same in adults and children in early life some variations may be observed in the duration and amplitude of the component parts of the electrocardiogram [40, 118]

**P Wave** The duration of the P wave in children should not exceed 0 09 second and the height 0 07 mm Rheumatic heart disease is the common cause of abnormal notching increase in height and widening of the P wave in younger patients Congenital heart disease particularly pulmonary stenosis may increase the height of the P wave but the other alterations are rarely encountered

**P R Intervals** According to Ashman the P R intervals from birth through the fifth year average 0 11 to 0 12 second from ages six to nine 0 12 second, from 10 to 12 0 135 second and for ages 13 and 14 0 14 sec

ond The normal P R intervals in children range from 0 14 to 0 18 second These variations depend on the age and the cardiac rate

**Right axis deviation** is a common finding at birth but tends to disappear in a few months (see Fig 188) Extreme right axis deviation is associated with congenital pulmonary stenosis while a less marked degree of right axis deviation accompanies mitral stenosis

**Left axis deviation** is not as common since the major causes of this shift in the axis are not present in children Coarctation of the aorta aortic regurgitation and stenosis and occasionally uncomplicated mitral regurgitation may produce left axis deviation

**The QRS complex** usually measures 0 06 to 0 08 second and never exceeds 0 09 second in normal records of children Low voltage of the QRS is rare A $Q_3$ wave is not abnormal in the presence of a right axis deviation

Occasionally bundle branch block may be encountered in healthy children The P R intervals in many of these cases are short (0 09 second) and paroxysms of tachycardia are often present An accessory A V bundle (bundle of Kent) has been offered as an explanation of this finding

**The RS T interval** may be elevated in all three leads in the presence of pericarditis in children as well as in adults This elevation must exceed 1 mm before it can be considered abnormal

**The T waves** in lead 1 are usually higher in children than in adults The height of T shows little variation However $T_3$ is not as high in children Congenital malformations tend to increase the height of the T waves

**Sinus arrhythmia** is common in children and is not abnormal Premature beats are rare but may accompany rheumatic heart disease Sometimes they may be observed in the absence of any other evidence of cardiac abnormality Prolongation of the P R interval is a common finding in the presence of rheumatic carditis although higher grades of block are rare Complete block may be congenital (see Case 54) In diphtheria complete heart block may occur and usually points to a poor prognosis Auricular fibrillation or flutter rarely occurs in children When present these arrhythmias complicate either rheumatic heart disease or cardiac failure secondary to a serious congenital defect (see Case 56)

## MISCELLANEOUS ELECTROCARDIOGRAPHIC PATTERNS

### Ventricular Strain

In addition to the great value of electrocardiography in the diagnosis of the various cardiac arrhythmias and acute myocardial infarction it has proved its usefulness in a variety of other conditions For example when either a sudden or chronic strain is placed upon either ventricle significant alterations appear In hypertension the T waves in lead 1 or in leads 1 and 2 may reflect the strain placed on the left ventricle long before the appear

ance of clinical signs of weakness or failure (Fig 250) If the strain is relieved the T waves return to normal (see Fig 121 A and B) It can also be shown by animal experimentation that a sudden right ventricular strain produced by temporarily clamping the pulmonary artery will cause a deep inversion of the T-wave in leads 2 and 3 Similar changes have been ob-

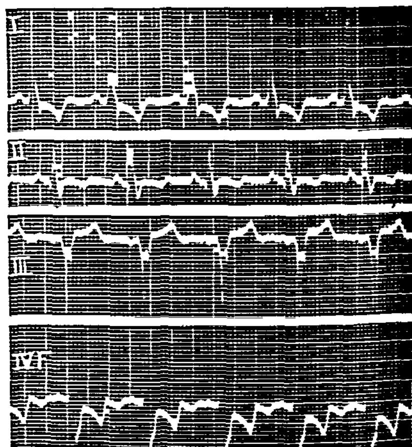



FIG. 250 Electrocardiogram taken on a woman of 6[illegible] with severe hypertension No digitalis had been given No history of angina or occlusion. Slight cardiac enlargement This tracing shows depressed S-T segments in leads I and 4F T is diphasic. [illegible] is suggestive of chronic left ventricular strain.

served in the human electrocardiogram following acute cor pulmonale (Fig 251) Consequently in many instances the electrocardiogram may distinguish between acute pulmonary embolism and sudden coronary occlusion. Ventricular strain produced by congenital lesions may also be reflected in the T waves of the electrocardiogram

Often the tracings reflecting ventricular strain resemble those produced by block of one of the bundle-branches Barnes[1] has [illegible] that th

pattern may be caused by metabolic changes produced by the excessive work placed upon the ventricular muscle Since the presence of fatigue products lessens conductivity and modifies electrical potential some change in the electrocardiogram would be expected

**Conditions that commonly place an extra load on the left ventricle** are hypertension aortic stenosis and aortic regurgitation As a result of this strain T wave changes and left axis deviation may be observed in the absence of any disease of the coronary arteries Occasionally in the presence of one of the conditions known to produce left ventricular strain signs of right ventricular strain may appear In these instances clinical evidence of mitral stenosis or pulmonary arteriosclerosis should be sought since either of these conditions may exert an influence on the right ventricle Consequently when the electrocardiogram displays an unexpected alteration the clinical evidence should be reviewed Often a satisfactory explanation will be found

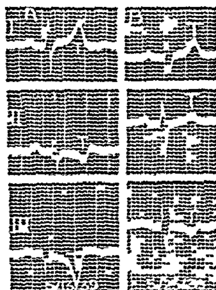


Fig 251 The electrocardiogram in acute right ventricular strain A was taken three hours after the sudden onset of chest pain and hemoptysis in a colored boy of 3 Note S wave in lead 1 the S-T take off below the isoelectric level in lead and 3 and small Q B was taken a week later Note the disappearance of the S wave in lead 1 upright T and decreased depth of T $Q_3$ has disappeared and there is a deep S wave in lead 3 The diagnosis of pulmonary embolism was made but its site of origin remained a mystery

Left ventricular strain first produces a left axis deviation The R wave in lead 1 is over 12 mm in height while $S_3$ is inverted and measures 5 mm or more below the base line If the strain continues the T wave in lead 1 may become inverted (Fig 250) T may show less marked inversion while $T_3$ has an opposite direction to $T_1$ Atypical tracings appear when other cardiac lesions such as pericarditis acute occlusion or mitral stenosis are present or follow ing the administration of digitalis

Occasionally in patients who show congestive failure secondary to hypertensive cardiovascular disease the blood pressure figure may be normal (page 105) In these instances the typical alterations in the electrocardiogram of left ventricular strain are valuable in suggesting the previous existence of hypertension

**Acute right ventricular strain** may be produced by pulmonary embolism the degree usually varying in proportion to the size of the artery occluded The S wave in lead 1 of the electrocardiogram becomes deeper

(Fig 251) T may be iso electric, inverted or diphasic If serial tracings are taken $T_1$ will be seen to again become upright $T_3$ is inverted, and a Q wave in lead 3 usually appears The RS T interval in lead 3 in many instances has a cove shape resembling that commonly produced by infarction It must be remembered that all of these changes may not appear in each case Moreover the occlusion of smaller branches of the pulmonary artery produces no electrocardiographic alteration consequently it is always possible for suggestive clinical symptoms to appear without change in the electrocardiogram Barnes [0] has shown that pulmonary infarction complicating the course of acute congestive failure seldom changes the electrocardiographic pattern to any extent, since the emboli in this condition are small and rarely occlude large pulmonary branches As a rule the *greater the degree of shock and circulatory embarrassment following a* pulmonary embolism the more likely it is that confirmatory evidence will appear in the electrocardiographic tracing The changes produced by pulmonary emboli are transient and tend to disappear from the electrocardiogram as soon as the right ventricle recovers from the effect of the sudden strain The exact cause of the production of these electrocardiographic changes is unknown Some observers attribute them to spasm of the coronary vessels of the affected ventricle Barnes believes that they are due to failure of a normal metabolic exchange in the muscle

Congenital pulmonary stenosis and some diseases of the pulmonary vessels and lungs lead to failure of the right ventricle Asthma associated with emphysema and pulmonary hypertension produces the same result Consequently the appearance of the pattern of right ventricular strain in the electrocardiogram in many of these cases comprises valuable additional information The typical electrocardiographic picture of this condition shows a deep S wave in lead 1 and a tall R wave in lead 3 (right axis deviation) $T_1$ is upright T may be positive but is more often diphasic or iso-electric while in long standing cases it is inverted $T_3$ is diphasic or inverted These T-wave changes may be produced by digitalis so it is important to make sure that the drug has not been administered before ascribing them to right ventricular strain

## Pericarditis

The diagnosis of pericarditis at the bedside presents many difficulties (Chapter 4) Consequently the recent recognition and description of electrocardiographic patterns characteristic of this condition have been great aids to the clinician The correct interpretation of these tracings however is only possible when the facts in each case are fully considered

The curves obtained in acute pericarditis resemble closely those produced by an area of infarction Often both conditions may be present simultaneously in which event the changes produced by the pericarditis are superimposed on those of the primary disease

When fluid is present in the pericardial sac it may exert pressure on the coronary vessels producing a myocardial ischemia that is reflected in

the T wave changes of the electrocardiogram[180] The low voltage of the waves of the tracing that is often observed may be caused by the effect of the fluid in the pericardial sac on the electrical impulses[8] Other observers[154, 377] believe that the elevation of the RS T segment of the electrocardiogram seen in acute pericarditis is the result of invasion of the subepicardial myocardium by the infectious process with the destruction of small areas of cardiac muscle It is quite likely that the latter change is the essential one in the production of the characteristic pattern observed in this disease (Figs 252 253)

In chronic pericarditis adhesions between the heart and the surrounding structures are unimportant When the layers of pericardium become thickened by scar tissue particularly when subsequently reinforced by calcium depositions the action of the heart may be seriously affected Diastolic filling is lessened Under these circumstances a change appears in the electrocardiogram (Fig 254) Low voltage of the QRS groups is the usual finding and as the condition progresses, this is often accompanied by flattening or inversion of the T waves in all leads

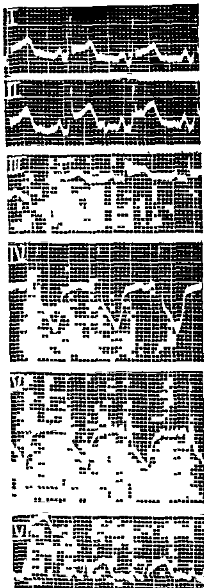



FIG 75 The electrocardiogram in acute pericarditis Note the elevation of the RS T segment in all indirect lead The direct lead in this tracing taken by the old technic (Courtesy of Dr Thomas McMillan )

### WOUNDS OF THE HEART

Electrocardiograms following penetrating wounds of the heart may show the presence of pericarditis produced by the incision the escape of blood into the pericardial sac or the infection introduced by the object causing the injury It is possible to distinguish between the electrocardiogram showing damage to the left coronary artery and the tracing produced when pericarditis is the only abnormality present When there is involvement of

the left coronary artery the RS T segment in lead 3 is depressed while the same interval is elevated in lead 1 (Fig 255) When this reciprocal relationship is absent injury to the coronary artery has usually not oc

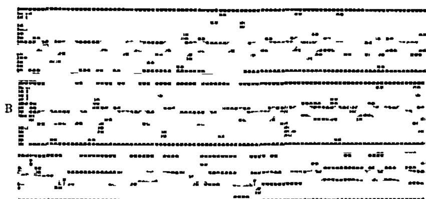


FIG 53 Electrocardiogram of patient of 26 taken during the first week of a febrile illness diagnosed grippe The heart was not enlarged and there were no joint manifestations Note slight elevation RS T segments in all leads The following day a friction rub appeared over the precordium

curred In many instances both effects combine to produce the electrocardiographic change (see Fig 159)

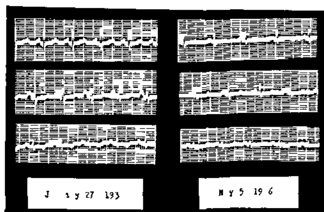



FIG 54 Electrocardiograms taken during the course of development of chronic constrictive (calcific) pericarditis Note change in voltage and flattening of T See Case No page 18

## DIABETES

The electrocardiographic pattern in diabetes is often influenced by the presence of coronary arteriosclerosis Insulin shock associated with hypoglycemia as well as diabetic coma and acidosis produce marked changes in the form of the electrocardiogram Flattened T waves of severe diabetics occasionally become upright and normal when appropriate treatment is in

stituted[94] During insulin shock associated with hypoglycemia the T waves in all leads are decreased in height and in some cases an increase in the A V conduction time has been noted [69]

Diabetic coma produces a depression of the S T interval of the electrocardiogram not unlike that observed following digitalis However in dia

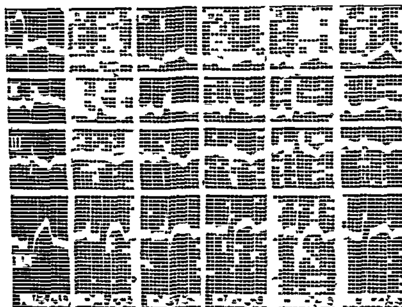


FIG 55 Serial electrocardiograms taken following a stab wound of the left ventricle (ice pick) Note elevation of the RS T segment in lead 1 of A and the reciprocal depression in lead 3 indicating involvement of the left coronary artery The series of tracings extend over a 10 day period during which time rapid return to normal may be seen Operation was not performed in this case

betic coma the Q T interval is prolonged while digitalis shortens this segment The electrocardiographic changes produced by diabetic coma are reversible Their cause is unknown

## MYXEDEMA

The electrocardiogram in myxedema (see Fig 142C) usually shows low voltage combined with flattening or inversion of the T waves Following treatment with thyroid gland the amplitude of all waves may return to normal Consequently in some cases the electrocardiogram may be of value in differentiating between the changes resulting from myxedema and those due to organic heart disease The characteristic form of the electrocardiogram in myxedema is not produced by alterations in the skin that characterize this condition but is most likely the result of actual myocardial change [1]

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